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(54) Title: MUTATIONS IN ION CHANNELS

(57) Abstract: A method of identifying a subject predisposed to a disorder associated with ion channel dysfunction, comprising ascertaining whether at least one of the genes encoding ion channel subunits in said subject has undergone a mutation event as set forth in one of SEQ ID Numbers: 1-72.



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MUTATIONS IN ION CHANNELSTechnical Field

The present invention is concerned with mutations in proteins having biological functions as ion channels and, more particularly, with such mutations where they are associated with diseases such as epilepsy and disorders associated with ion channel dysfunction including, but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness.

Background Art

Epilepsies constitute a diverse collection of brain disorders that affect about 3% of the population at some time in their lives (Annegers, 1996). An epileptic seizure can be defined as an episodic change in behaviour caused by the disordered firing of populations of neurons in the central nervous system. This results in varying degrees of involuntary muscle contraction and often a loss of consciousness. Epilepsy syndromes have been classified into more than 40 distinct types based upon characteristic symptoms, types of seizure, cause, age of onset and EEG patterns (Commission on Classification and Terminology of the International League Against Epilepsy, 1989). However the single feature that is common to all syndromes is the persistent increase in neuronal excitability that is both occasionally and unpredictably expressed as a seizure.

A genetic contribution to the aetiology of epilepsy has been estimated to be present in approximately 40% of

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affected individuals (Gardiner, 2000). As epileptic seizures may be the end-point of a number of molecular aberrations that ultimately disturb neuronal synchrony, the genetic basis for epilepsy is likely to be heterogeneous. There are over 200 Mendelian diseases which include epilepsy as part of the phenotype. In these diseases, seizures are symptomatic of underlying neurological involvement such as disturbances in brain structure or function. In contrast, there are also a number of "pure" epilepsy syndromes in which epilepsy is the sole manifestation in the affected individuals. These are termed idiopathic and account for over 60% of all epilepsy cases.

Idiopathic epilepsies have been further divided into partial and generalized sub-types. Partial (focal or local) epileptic fits arise from localized cortical discharges, so that only certain groups of muscles are involved and consciousness may be retained. However, in generalized epilepsy, EEG discharge shows no focus such that all subcortical regions of the brain are involved. Although the observation that generalized epilepsies are frequently inherited is understandable, the mechanism by which genetic defects, presumably expressed constitutively in the brain, give rise to partial seizures is less clear.

The molecular genetic era has resulted in spectacular advances in classification, diagnosis and biological understanding of numerous inherited neurological disorders including muscular dystrophies, familial neuropathies and spinocerebellar degenerations. These disorders are all uncommon or rare and have simple Mendelian inheritance. In contrast, common neurological diseases like epilepsy, have complex inheritance where they are determined by multiple genes sometimes interacting with environmental influences. Molecular genetic advances in disorders with complex inheritance have been far more modest to date (Todd, 1999).

Most of the molecular genetic advances have occurred by a sequential three stage process. First a clinically homogeneous disorder is identified and its mode of inheritance determined. Second, linkage analysis is performed on carefully characterized clinical populations with the disorder. Linkage analysis is a process where the chromosomal localization of a particular disorder is narrowed down to approximately 0.5% of the total genome. Knowledge of linkage imparts no intrinsic biological insights other than the important clue as to where to look in the genome for the abnormal gene. Third, strategies such as positional cloning or the positional candidate approach are used to identify the aberrant gene and its specific mutations within the linked region (Collins, 1995).

Linkage studies in disorders with complex inheritance have been bedevilled by negative results and by failure to replicate positive findings. A sense of frustration permeates current literature in the genetics of complex disorders. Carefully performed, large scale studies involving hundreds of sibpairs in disorders including multiple sclerosis and diabetes have been essentially negative (Bell and Lathrop, 1996; Lernmark and Ott, 1998). An emerging view is that such disorders are due to the summation of many genes of small effect and that identification of these genes may only be possible with very large-scale association studies. Such studies on a genome-wide basis are currently impossible due to incomplete marker sets and computational limitations.

The idiopathic generalized epilepsies (IGE) are the most common group of inherited human epilepsy and do not have simple inheritance. Like other complex disorders, linkage studies in IGE have generated controversial and conflicting claims. Previous authors have suggested the possibility of multifactorial, polygenic, oligogenic or two locus models for the disease (Andermann, 1982; Doose



and Baier, 1989; Greenberg et al., 1988a; 1992; Janz et al., 1992).

Two broad groups of IGE are now known - the classical idiopathic generalized epilepsies (Commission on  
5 Classification and Terminology of the International League Against Epilepsy, 1989) and the newly recognized genetic syndrome of generalized epilepsy with febrile seizures plus (GEFS<sup>+</sup>) (Scheffer and Berkovic, 1997; Singh et al., 1999).

10 The classical IGEs are divided into a number of clinically recognizable but overlapping sub-syndromes including childhood absence epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy etc (Commission on  
15 Classification and Terminology of the International League Against Epilepsy, 1989; Roger et al., 1992). The sub-syndromes are identified by age of onset and the pattern of seizure types (absence, myoclonus and tonic-clonic). Some patients, particularly those with tonic-clonic  
20 seizures alone do not fit a specifically recognized sub-syndrome. Arguments for regarding these as separate syndromes, yet recognizing that they are part of a neurobiological continuum, have been presented previously (Berkovic et al. 1987; 1994; Reutens and Berkovic, 1995).

GEFS<sup>+</sup> was originally recognized through large multi-  
25 generation families and comprises a variety of sub-syndromes. Febrile seizures plus (FS<sup>+</sup>) is a sub-syndrome where children have febrile seizures occurring outside the age range of 3 months to 6 years, or have associated febrile tonic-clonic seizures. Many family members have a  
30 phenotype indistinguishable from the classical febrile convulsion syndrome and some have FS<sup>+</sup> with additional absence, myoclonic, atonic, or complex partial seizures. The severe end of the GEFS<sup>+</sup> spectrum includes myoclonic-astatic epilepsy.

35 The cumulative incidence for epilepsy by age 30 years (proportion suffering from epilepsy at some time) is 1.5% (Hauser et al., 1993). Accurate estimates for the

cumulative incidence of the IGEs are unavailable. In epidemiological studies where attempts are made to subclassify epilepsies, rather few cases of IGE are diagnosed, and many cases are unclassified. This is probably because cases are rarely directly examined by epileptologists. In clinic- or office-based series seen by experts, most cases are classifiable and IGEs account for about 25% of cases. This suggests that about 0.3% of the population suffer from IGE at some time.

In outbred populations many patients with classical IGE appear to be sporadic as siblings and parents are usually unaffected. Systematic EEG studies on clinically unaffected family members show an increase in age-dependent occurrence of generalized epileptiform discharges compared to controls. In addition, to the approximate 0.3% of the population with clinical IGE, systematic EEG studies suggest that about 1% of healthy children have generalized epileptiform discharges while awake (Cavazzuti et al., 1980; Okubo et al., 1994).

Approximately 5-10% of first degree relatives of classical IGE probands have seizures with affected relatives usually having IGE phenotypes or febrile seizures. While nuclear families with 2-4 affected individuals are well recognized and 3 generation families or grandparent-grandchild pairs are occasionally observed (Italian League Against Epilepsy Genetic Collaborative Group, 1993), families with multiple affected individuals extending over 4 or more generations are exceptionally rare.

For GEFS<sup>+</sup>, however, a number of large multi-generation families showing autosomal dominant inheritance with incomplete penetrance are known. Similar to classical IGE, analysis of sporadic cases and small families with GEFS<sup>+</sup> phenotypes does not suggest simple Mendelian inheritance. Indeed, bilineal inheritance, where there is a history of epilepsy on maternal and paternal sides, is observed in both GEFS<sup>+</sup> and classical IGE families (Singh et al., 1999;

Italian League Against Epilepsy Genetic Collaborative Group, 1993).

Within single families with classical IGE or GEFS<sup>+</sup>, affected individuals often have different sub-syndromes. The closer an affected relative is to the proband, the more similar are their sub-syndromes, and siblings often have similar sub-syndromes (Italian League Against Epilepsy Genetic Collaborative Group, 1993). Less commonly, families are observed where most, or all, known affected individuals have one classical IGE sub-syndrome such as childhood absence epilepsy or juvenile myoclonic epilepsy (Italian League Against Epilepsy Genetic Collaborative Group, 1993).

Importantly, sub-syndromes are identical in affected monozygous twins with IGE. In contrast, affected dizygous twins, may have the same or different sub-syndromes. Classical IGE and GEFS<sup>+</sup> sub-syndromes tend to segregate separately (Singh et al., 1999).

In some inbred communities, pedigree analysis strongly suggests recessive inheritance for juvenile myoclonic epilepsy and other forms of IGE (Panayiotopoulos and Obeid, 1989; Berkovic et al., 2000). In such families, sub-syndromes are much more similar in affected siblings than in affected sib-pairs from outbred families. Recently, a family with an infantile form of IGE with autosomal recessive inheritance, confirmed by linkage analysis, was described in Italy (Zara et al., 2000).

Most work on the molecular genetics of classical IGEs has been done on the sub-syndrome of juvenile myoclonic epilepsy where a locus in proximity or within the HLA region on chromosome 6p was first reported in 1988 (Greenberg et al., 1988b). This finding was supported by two collaborating laboratories, in separate patient samples, and subsequently three groups provided further evidence for a 6p locus for juvenile myoclonic epilepsy in some, but not all, of their families. However, genetic defects have not been found and the exact locus of the

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gene or genes, in relationship to the HLA region, remains controversial. Strong evidence for linkage to chromosome 6 also comes from a study of a single large family with juvenile myoclonic epilepsy, but in this pedigree the locus is well outside the HLA region. A locus on chromosome 15q has also been suggested for juvenile myoclonic epilepsy, but was not confirmed by two other studies.

In general, the results of studies of the putative chromosomal 6p locus in the HLA region in patients with absence epilepsies or other forms of idiopathic generalized epilepsies have been negative. The major exception is that study of probands with tonic-clonic seizures on awakening, a sub-syndrome closely related to juvenile myoclonic epilepsy, suggests linkage to 6p.

Linkage for classical remitting childhood absence epilepsy remains elusive, but in a family with persisting absence evolving into a juvenile myoclonic epilepsy phenotype, linkage to chromosome 1p has been claimed. An Indian pedigree with persisting absence and tonic-clonic seizures may link to 8q24. Linkage to this region was also suggested by a non-parametric analysis in IGE, irrespective of subsyndrome, but was not confirmed in another study. Other loci for IGEs that have been reported in single studies include 3p14, 8p, 18 and possibly 5p. The unusual example of recessively inherited infantile onset IGE described in Italy maps to 16p in a single family.

Thus, like most disorders with complex inheritance, the literature on genetics of classical IGEs is confusing and contradictory. Some, and perhaps much, of this confusion is due to heterogeneity, with the likelihood of a number of loci for IGEs. The studies reviewed above were principally performed on multiple small families, so heterogeneity within and between samples is probable. Whether all, some, or none of the linkages reported above will be found to harbour relevant genes for IGE remains to

be determined. Most of the studies reviewed above used analysis methods assuming Mendelian inheritance, an assumption that is not correct for outbred communities. Some studies used multiple models (autosomal recessive, autosomal dominant). Although parametric linkage analysis may be reliable in some circumstance of analyzing complex disease, it can lead to spurious findings as highlighted by the literature on linkage in major psychoses (Risch and Botstein, 1996).

In so far as GEFS<sup>+</sup> is concerned, linkage analysis on rare multi-generation large families with clinical evidence of a major autosomal dominant gene have demonstrated loci on chromosomes 19q and 2q. Both the 19q and 2q GEFS<sup>+</sup> loci have been confirmed in independently ascertained large families, and genetic defects have been identified. Families linked to 19q are known and a mutation in the gene for the  $\beta 1$  subunit of the neuronal sodium channel (SCN1B) has been identified (Wallace et al., 1998). This mutation results in the loss of a critical disulphide bridge of this regulatory subunit and causes a loss of function *in vitro*. Families linked to 2q are also known and mutations in the pore-forming  $\alpha$  subunit of the neuronal sodium channel (SCN1A) have been identified (PCT/AU01/01648; Wallace et al., 2001b; Escayg et al., 2000). Studies on the more common small families with GEFS<sup>+</sup> have not revealed these or other mutations to date.

In addition to the SCN1B and SCN1A mutations in GEFS<sup>+</sup>, four other gene defects have been discovered for human idiopathic epilepsies through the study of large families. Mutations in the alpha-4 subunit of the neuronal nicotinic acetylcholine receptor (CHRNA4) occur in the focal epilepsy syndrome of autosomal dominant nocturnal frontal lobe epilepsy (Australian patent AU-B-56247/96; Steinlein et al., 1995). Mutations in the gamma-2 subunit of the GABA<sub>A</sub> receptor (GABRG2) have been identified in childhood absence epilepsy, febrile seizures (including febrile

seizures plus) and myoclonic epilepsy (PCT/AU01/00729; Wallace et al., 2001a). Finally, mutations in two potassium channel genes (KCNQ2 and KCNQ3) were identified in benign familial neonatal convulsions (Singh et al., 1998; Biervert et al., 1998; Charlier et al., 1998). Although initially regarded as a special form of IGE, this unusual syndrome is probably a form of inherited focal epilepsy.

Further to these studies, mutations in other genes have been identified to be causative of epilepsy. These include mutations in the beta-2 subunit (CHRNA2) of the neuronal nicotinic acetylcholine receptor (PCT/AU01/00541; Phillips et al., 2001) and the delta subunit (GABRD) of the GABA<sub>A</sub> receptor (PCT/AU01/00729).

A number of mouse models approximating human IGE are known. These mice mutants have ataxia in addition to generalized spike-and-wave discharges with absences or tonic-clonic seizures. Recessive mutations in calcium channel subunit genes have been found in lethargic (CACNB4), tottering/leaner (CACNA1A), and stargazer (CACNG2) mutants. The slow-wave epilepsy mouse mutant has a mutation in the sodium/hydrogen exchanger gene, which may have important downstream effects on pH-sensitive ion channels.

The human and mouse literature is now suggesting that the idiopathic epilepsies comprise a family of channelopathies with mutations in ion channel subunits of voltage-gated (eg SCN1A, SCN1B, KCNQ2, KCNQ3) or ligand-gated (eg CHRNA4, CHRNA2, GABRG2, GABRD) types. These channels are typically comprised of a number of subunits, specified by genes on different chromosomes. The stoichiometry and conformation of ion channel subunits are not yet well understood, but many have multiple subunits in a variety of combinations.

The involvement of ion channels in other neuro/physiological disorders has also been observed (reviewed in Dworakowska and Dolowy, 2000). Mutations in

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voltage-gated sodium, potassium, calcium and chloride channels as well as ligand-gated channels such as the acetylcholine and GABA receptors may lead to physiological disorders such as hyper- and hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia and cardiac arrhythmias. Neurological disorders other than epilepsy that are associated with ion channel mutations include episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, as well as neuropathic pain, inflammatory pain and chronic/acute pain. Some kidney disorders such as Bartter's syndrome, polycystic kidney disease and Dent's disease, secretion disorders such as hyperinsulinemic hypoglycemia of infancy and cystic fibrosis, and vision disorders such as congenital stationary night blindness and total colour-blindness may also be linked to mutations in ion channels.

#### Disclosure of the Invention

In a new genetic model for the idiopathic generalised epilepsies (IGEs) described in PCT/AU01/00872 (the disclosure of which is incorporated herein by reference) it has been postulated that most classical IGE and GEFS<sup>+</sup> cases are due to the combination of two mutations in multi-subunit ion channels. These are typically point mutations resulting in a subtle change of function. The critical postulate is that two mutations, usually, but not exclusively, in different subunit alleles ("digenic model"), are required for clinical expression of IGE. It was further proposed that

a) A number of different mutated subunit pairs can be responsible for IGE. Combinations of two mutated subunits lead to an IGE genotype with ~30% penetrance.

b) The total allele frequency of mutated subunits is ~8%. It was calculated that approximately 15% of the population has one or more mutated

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subunit genes and 1% have two or more mutated subunits.

5 c) Sub-syndromes are principally determined by the specific combination of mutated subunit pairs, although one or more other genes, including ion channel subunits, of smaller effect may modify the phenotype.

10 d) Mutated subunit combinations that cause classical IGEs are largely separate from those that cause GEFS<sup>+</sup>, although some subunits may be involved in both syndromes.

15 e) Individuals with single 'change of function' mutations would not have IGE, but such mutations may contribute to simple febrile seizures, which are observed with increased frequency in relatives of IGE probands.

The model also proposes that subunit mutations with more severe functional consequences (eg breaking a disulphide bridge in SCN1B or amino acid substitution in the pore forming regions of SCN1A for GEFS<sup>+</sup>) cause autosomal dominant generalized epilepsies with a penetrance of 60-90%. The precise sub-syndromes in GEFS<sup>+</sup> are determined by minor allelic variation or mutations in other ion channel subunits. Such "severe" mutations are rare (allele frequency <0.01%) and are infrequent causes of GEFS<sup>+</sup>. They very rarely, or perhaps never, cause classical IGE.

30 The identification of molecular changes in ion channel subunits is therefore a significant step towards the elucidation of genetic variants that alone or in combination (based on the digenic model) give rise to an epilepsy phenotype, and to other neuro/physiological disorders associated with ion channel dysfunction.

35 The present inventors have identified a number of novel mutations or variants in genes encoding subunits of ion channels in individuals with epilepsy. It will be appreciated that for each molecular defect one can provide



an isolated nucleic acid molecule coding for a protein having a biological function as part of an ion channel in a mammal, wherein a mutation event selected from the group consisting of point mutations, deletions, insertions and rearrangements has occurred so as to affect the functioning of the ion channel. In some instances this single mutation alone will produce a phenotype of epilepsy or other neuro/physiological disorders associated with ion channel dysfunction.

In the case where a single mutation alone does not produce, say, an epilepsy phenotype, there would be provided one or more additional isolated nucleic acid molecules coding for proteins having a biological function as part of an ion channel in a mammal, wherein a mutation event selected from the group consisting of point mutations, deletions, insertions and rearrangements has occurred so as to affect the functioning of the ion channel. The cumulative effect of the mutations in each isolated nucleic acid molecule *in vivo* is to produce a epilepsy or another neuro/physiological disorders in said mammal. The mutations may be in nucleic acid molecules coding for protein subunits belonging to the same ion channel or may be in nucleic acid molecules coding for protein subunits that belong to different ion channels.

Typically such mutations are point mutations and the ion channels are voltage-gated channels such as a sodium, potassium, calcium or chloride channels or are ligand-gated channels such as members of the nAChR/GABA super family of receptors, or a functional fragment or homologue thereof.

Mutations may include those in non-coding regions of the ion channel subunits (eg mutations in the promoter region which affect the level of expression of the subunit gene, mutations in intronic sequences which affect the correct splicing of the subunit during mRNA processing, or mutations in the 5' or 3' untranslated regions that can affect translation or stability of the mRNA). Mutations

may also and more preferably will be in coding regions of the ion channel subunits (eg nucleotide mutations may give rise to an amino acid change in the encoded protein or nucleotide mutations that do not give rise to an amino acid change but may affect the stability of the mRNA).

Mutation combinations may be selected from, but are not restricted to, those identified in Table 1.

Accordingly in one aspect of the present invention there is provided a method of identifying a subject predisposed to a disorder associated with ion channel dysfunction, comprising ascertaining whether at least one of the genes encoding ion channel subunits in said subject has undergone a mutation event selected from the group consisting of the mutation events set forth in the following Table:

Subunit Gene	Exon/Intron	DNA Mutation
SCN1A	Exon 5	c664C→T
SCN1A	Exon 8	c1152G→A
SCN1A	Exon 9	c1183G→C
SCN1A	Exon 9	c1207T→C
SCN1A	Exon 9	c1237T→A
SCN1A	Exon 9	c1265T→A
SCN1A	Exon 21	c4219C→T
SCN1A	Exon 26	c5339T→C
SCN1A	Exon 26	c5674C→T
SCN1B	Exon 3	c254G→A
SCN2A	Exon 6A	c668G→A
SCN2A	Exon 16	c2674G→A
SCN2A	Exon 17	c3007C→A
SCN2A	Exon 19	c3598A→G
SCN2A	Exon 20	c3956G→A
SCN2A	Exon 12	c1785T→C
SCN2A	Exon 27	c4919T→A
SCN1A	Intron 9	IVS9-1G→A
SCN1A	Intron 23	IVS23+33G→A
SCN2A	Intron 7	IVS7+61T→A
SCN2A	Intron 19	IVS19-55A→G
SCN2A	Intron 22	IVS22-31A→G
SCN2A	Intron 2	IVS2-28G→A
SCN2A	Intron 8	IVS8-3T→C
SCN2A	Intron 11	IVS11+49A→G
SCN2A	Intron 11	IVS11-16C→T

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SCN2A	Intron 17	IVS17-71C→T
SCN2A	Intron 17	IVS17-74delG
SCN2A	Intron 17	IVS17-74insG
CHRNA5	Exon 4	c400G→A
CHRNA2	Exon 4	c373G→A
CHRNA3	Exon 2	c110G→A
CHRNA2	Exon 4	c351C→T
CHRNA2	Exon 5	c771C→T
CHRNA3	Exon 2	c159A→G
CHRNA3	Exon 4	c291G→A
CHRNA3	Exon 4	c345G→A
CHRNA2	Intron 3	IVS3-16C→T
CHRNA3	Intron 3	IVS3-5T→C
CHRNA3	Intron 4	IVS4+8G→C
KCNQ2	Exon 1	c204-c205insC
KCNQ2	Exon 1	c1A→G
KCNQ2	Exon 1	c2T→C
KCNQ2	Exon 8	c1057C→G
KCNQ2	Exon 11	c1288C→T
KCNQ2	Exon 14	c1710A→T
KCNQ2	Exon 15	c1856T→G
KCNQ2	Intron 9	IVS9+(46-48)delCCT
KCNQ3	Intron 11	IVS11+43G→A
KCNQ3	Intron 12	IVS12+29G→A
GABRB1	Exon 5	c508C→T
GABRB1	Exon 9	c1329G→A
GABRB1	Exon 8	c975C→T
GABRG3	Exon 8	c995T→C
GABRA1	5' UTR	c-142A→G
GABRA1	5' UTR	c-31C→T
GABRA2	3' UTR	c1615G→A
GABRA5	5' UTR	c-271G→C
GABRA5	5' UTR	c-228A→G
GABRA5	5' UTR	c-149G→C
GABRB2	5' UTR	c-159C→T
GABRB2	3' UTR	c1749C→T
GABRPi	5' UTR	c-101C→T
GABRB1	Intron 1	IVS1+24T→G
GABRB1	Intron 6	IVS6+72T→G
GABRB1	Intron 7	IVS7-34A→G
GABRB3	Intron 1	IVS1-14C→T
GABRB3	Intron 7	IVS7+58delAA
GABRD	Intron 6	IVS6+132insC
GABRD	Intron 6	IVS6+130insC
GABRD	Intron 6	IVS6+73delCGCGCCACCGCCCTTCCGCG
GABRG3	Intron 8	IVS8-102C→T

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In a further aspect there is provided a method of identifying a subject predisposed to a disorder associated with ion channel dysfunction, comprising ascertaining whether at least one of the genes encoding ion channel subunits in said subject has undergone a mutation event as set forth in one of SEQ ID Numbers: 1-72.

In another aspect of the present invention there is provided an isolated nucleic acid molecule encoding a mutant or variant ion channel subunit wherein a mutation event selected from the group consisting of the mutation events set forth in the following Table:

Subunit Gene	Exon/Intron	DNA Mutation
SCN1A	Exon 5	c664C→T
SCN1A	Exon 8	c1152G→A
SCN1A	Exon 9	c1183G→C
SCN1A	Exon 9	c1207T→C
SCN1A	Exon 9	c1237T→A
SCN1A	Exon 9	c1265T→A
SCN1A	Exon 21	c4219C→T
SCN1A	Exon 26	c5339T→C
SCN1A	Exon 26	c5674C→T
SCN1B	Exon 3	c254G→A
SCN2A	Exon 6A	c668G→A
SCN2A	Exon 16	c2674G→A
SCN2A	Exon 17	c3007C→A
SCN2A	Exon 19	c3598A→G
SCN2A	Exon 20	c3956G→A
SCN2A	Exon 12	c1785T→C
SCN2A	Exon 27	c4919T→A
SCN1A	Intron 9	IVS9-1G→A
SCN1A	Intron 23	IVS23+33G→A
SCN2A	Intron 7	IVS7+61T→A
SCN2A	Intron 19	IVS19-55A→G
SCN2A	Intron 22	IVS22-31A→G
SCN2A	Intron 2	IVS2-28G→A
SCN2A	Intron 8	IVS8-3T→C
SCN2A	Intron 11	IVS11+49A→G
SCN2A	Intron 11	IVS11-16C→T
SCN2A	Intron 17	IVS17-71C→T
SCN2A	Intron 17	IVS17-74delG
SCN2A	Intron 17	IVS17-74insG
CHRNA5	Exon 4	c400G→A
CHRNA2	Exon 4	c373G→A

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CHRNA3	Exon 2	c110G→A
CHRNA2	Exon 4	c351C→T
CHRNA2	Exon 5	c771C→T
CHRNA3	Exon 2	c159A→G
CHRNA3	Exon 4	c291G→A
CHRNA3	Exon 4	c345G→A
CHRNA2	Intron 3	IVS3-16C→T
CHRNA3	Intron 3	IVS3-5T→C
CHRNA3	Intron 4	IVS4+8G→C
KCNQ2	Exon 1	c204-c205insC
KCNQ2	Exon 1	c1A→G
KCNQ2	Exon 1	c2T→C
KCNQ2	Exon 8	c1057C→G
KCNQ2	Exon 11	c1288C→T
KCNQ2	Exon 14	c1710A→T
KCNQ2	Exon 15	c1856T→G
KCNQ2	Intron 9	IVS9+(46-48) delCCT
KCNQ3	Intron 11	IVS11+43G→A
KCNQ3	Intron 12	IVS12+29G→A
GABRB1	Exon 5	c508C→T
GABRB1	Exon 9	c1329G→A
GABRB1	Exon 8	c975C→T
GABRG3	Exon 8	c995T→C
GABRA1	5' UTR	c-142A→G
GABRA1	5' UTR	c-31C→T
GABRA2	3' UTR	c1615G→A
GABRA5	5' UTR	c-271G→C
GABRA5	5' UTR	c-228A→G
GABRA5	5' UTR	c-149G→C
GABRB2	5' UTR	c-159C→T
GABRB2	3' UTR	c1749C→T
GABRPi	5' UTR	c-101C→T
GABRB1	Intron 1	IVS1+24T→G
GABRB1	Intron 6	IVS6+72T→G
GABRB1	Intron 7	IVS7-34A→G
GABRB3	Intron 1	IVS1-14C→T
GABRB3	Intron 7	IVS7+58delAA
GABRD	Intron 6	IVS6+132insC
GABRD	Intron 6	IVS6+130insC
GABRD	Intron 6	IVS6+73delCGCGCCCACCGCCCCTTCCGCG
GABRG3	Intron 8	IVS8-102C→T

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has occurred.

In still another aspect of the present invention there is provided an isolated nucleic acid molecule encoding a mutant or variant ion channel subunit wherein a

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mutation event has occurred as set forth in one of SEQ ID Numbers: 1-72.

The mutation event disrupts the functioning of an ion channel so as to produce a phenotype of epilepsy, and/or one or more other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness, either alone or in combination with one or more additional mutations or variations in the ion channel subunit genes.

In another aspect of the present invention there is provided an isolated nucleic acid molecule encoding a mutant KCNQ2 subunit, wherein the mutation event has occurred in the C-terminal domain of the KCNQ2 subunit and leads to a disturbance in the calmodulin binding affinity of the subunit, so as to produce an epilepsy phenotype.

In one form of the invention, the mutations are in exon 8 or exon 15 of the KCNQ2 subunit and result in the replacement of an arginine residue with a glycine residue at amino acid position 353, or the replacement of a leucine residue with an arginine at amino acid position 619. The R353G mutation occurs as a result of a C to G nucleotide substitution at position 1057 of the KCNQ2 coding sequence as shown in SEQ ID NO: 44. The L619R mutation occurs as a result of a T to G nucleotide substitution at position 1856 of the KCNQ2 coding sequence as shown in SEQ ID NO: 47.

In a further form of the invention, the mutations are in exon 11 or exon 14 of the KCNQ2 subunit and result in

the replacement of an arginine residue with a stop codon at amino acid position 430, or the replacement of an arginine residue with a serine at amino acid position 570. The R430X mutation occurs as a result of a C to T nucleotide substitution at position 1288 of the KCNQ2 coding sequence as shown in SEQ ID NO: 45. The R570S mutation occurs as a result of an A to T nucleotide substitution at position 1710 of the KCNQ2 coding sequence as shown in SEQ ID NO: 46.

Preferably these mutations create a phenotype of benign familial neonatal seizures (BFNS).

In a further aspect of the present invention there is provided a combination of two or more isolated nucleic acid molecules each having a novel mutation event as laid out in Table 1. The cumulative effect of the mutations in each isolated nucleic acid molecule *in vivo* is to produce an epilepsy or another disorder associated with ion channel dysfunction as described above in said mammal.

In a particularly preferred embodiment of the present invention, the isolated nucleic acid molecules have a nucleotide sequence as shown in any one of SEQ ID Numbers: 1-72. The sequences correspond to the novel DNA mutations or variants laid out in Table 1.

In another aspect of the present invention there is provided an isolated nucleic acid molecule comprising any one of the nucleotide sequences set forth in SEQ ID Numbers: 1-72.

In another aspect of the present invention there is provided an isolated nucleic acid molecule consisting of any one of the nucleotide sequences set forth in SEQ ID Numbers: 1-72.

The nucleotide sequences of the present invention can be engineered using methods accepted in the art for a variety of purposes. These include, but are not limited to, modification of the cloning, processing, and/or expression of the gene product. PCR reassembly of gene fragments and the use of synthetic oligonucleotides allow

the engineering of the nucleotide sequences of the present invention. For example, oligonucleotide-mediated site-directed mutagenesis can introduce further mutations that create new restriction sites, alter expression patterns and produce splice variants etc.

As a result of the degeneracy of the genetic code, a number of polynucleotide sequences, some that may have minimal similarity to the polynucleotide sequences of any known and naturally occurring gene, may be produced. Thus, the invention includes each and every possible variation of a polynucleotide sequence that could be made by selecting combinations based on possible codon choices. These combinations are made in accordance with the standard triplet genetic code as applied to the polynucleotide sequences of the present invention, and all such variations are to be considered as being specifically disclosed.

The nucleic acid molecules of this invention are typically DNA molecules, and include cDNA, genomic DNA, synthetic forms, and mixed polymers, both sense and antisense strands, and may be chemically or biochemically modified, or may contain non-natural or derivatised nucleotide bases as will be appreciated by those skilled in the art. Such modifications include labels, methylation, intercalators, alkylators and modified linkages. In some instances it may be advantageous to produce nucleotide sequences possessing a substantially different codon usage than that of the polynucleotide sequences of the present invention. For example, codons may be selected to increase the rate of expression of the peptide in a particular prokaryotic or eukaryotic host corresponding with the frequency that particular codons are utilized by the host. Other reasons to alter the nucleotide sequence without altering the encoded amino acid sequences include the production of RNA transcripts having more desirable properties, such as a greater half-



life, than transcripts produced from the naturally occurring mutated sequence.

The invention also encompasses production of nucleic acid sequences of the present invention entirely by synthetic chemistry. Synthetic sequences may be inserted into expression vectors and cell systems that contain the necessary elements for transcriptional and translational control of the inserted coding sequence in a suitable host. These elements may include regulatory sequences, promoters, 5' and 3' untranslated regions and specific initiation signals (such as an ATG initiation codon and Kozak consensus sequence) which allow more efficient translation of sequences encoding the polypeptides of the present invention. In cases where the complete coding sequence, including the initiation codon and upstream regulatory sequences, are inserted into the appropriate expression vector, additional control signals may not be needed. However, in cases where only coding sequence, or a fragment thereof, is inserted, exogenous translational control signals as described above should be provided by the vector. Such signals may be of various origins, both natural and synthetic. The efficiency of expression may be enhanced by the inclusion of enhancers appropriate for the particular host cell system used (Scharf et al., 1994).

The invention also includes nucleic acid molecules that are the complements of the sequences described herein.

The present invention allows for the preparation of purified polypeptide or protein from the polynucleotides of the present invention, or variants thereof. In order to do this, host cells may be transformed with a novel nucleic acid molecule as described above, or with nucleic acid molecules encoding two or more mutant ion channel subunits. If the mutant subunits form a part of the same ion channel a receptor protein containing two or more mutant subunits may be isolated. If the mutant subunits are subunits of different ion channels the host cells will

express two or more mutant receptor proteins. Typically said host cells are transfected with an expression vector comprising a DNA molecule according to the invention or, in particular, DNA molecules encoding two or more mutant ion channel subunits. A variety of expression vector/host systems may be utilized to contain and express sequences encoding polypeptides of the invention. These include, but are not limited to, microorganisms such as bacteria transformed with plasmid or cosmid DNA expression vectors; yeast transformed with yeast expression vectors; insect cell systems infected with viral expression vectors (e.g., baculovirus); or mouse or other animal or human tissue cell systems. Mammalian cells can also be used to express a protein using a vaccinia virus expression system. The invention is not limited by the host cell or vector employed.

The polynucleotide sequences, or variants thereof, of the present invention can be stably expressed in cell lines to allow long term production of recombinant proteins in mammalian systems. Sequences encoding the polypeptides of the present invention can be transformed into cell lines using expression vectors which may contain viral origins of replication and/or endogenous expression elements and a selectable marker gene on the same or on a separate vector. The selectable marker confers resistance to a selective agent, and its presence allows growth and recovery of cells which successfully express the introduced sequences. Resistant clones of stably transformed cells may be propagated using tissue culture techniques appropriate to the cell type.

The protein produced by a transformed cell may be secreted or retained intracellularly depending on the sequence and/or the vector used. As will be understood by those of skill in the art, expression vectors containing polynucleotides which encode a protein may be designed to contain signal sequences which direct secretion of the protein through a prokaryotic or eukaryotic cell membrane.

In addition, a host cell strain may be chosen for its ability to modulate expression of the inserted sequences or to process the expressed protein in the desired fashion. Such modifications of the polypeptide include, but are not limited to, acetylation, glycosylation, phosphorylation, and acylation. Post-translational cleavage of a "prepro" form of the protein may also be used to specify protein targeting, folding, and/or activity. Different host cells having specific cellular machinery and characteristic mechanisms for post-translational activities (e.g., CHO or HeLa cells), are available from the American Type Culture Collection (ATCC) and may be chosen to ensure the correct modification and processing of the foreign protein.

When large quantities of the protein product of the gene are needed, such as for antibody production, vectors which direct high levels of expression of this protein may be used, such as those containing the T5 or T7 inducible bacteriophage promoter. The present invention also includes the use of the expression systems described above in generating and isolating fusion proteins which contain important functional domains of the protein. These fusion proteins are used for binding, structural and functional studies as well as for the generation of appropriate antibodies.

In order to express and purify the protein as a fusion protein, the appropriate cDNA sequence is inserted into a vector which contains a nucleotide sequence encoding another peptide (for example, glutathionine succinyl transferase). The fusion protein is expressed and recovered from prokaryotic or eukaryotic cells. The fusion protein can then be purified by affinity chromatography based upon the fusion vector sequence. The desired protein is then obtained by enzymatic cleavage of the fusion protein.

Fragments of the polypeptides of the present invention may also be produced by direct peptide synthesis

using solid-phase techniques. Automated synthesis may be achieved by using the ABI 431A Peptide Synthesizer (Perkin-Elmer). Various fragments of this protein may be synthesized separately and then combined to produce the full-length molecule.

The present invention is also concerned with polypeptides having a biological function as an ion channel in a mammal, wherein a mutation event selected from the group consisting of substitutions, deletions, truncations, insertions and rearrangements has occurred so as to affect the functioning of the ion channel. In some instances this single mutation alone will produce an epilepsy phenotype or other neuro/physiological disorders associated with ion channel dysfunction.

In the case where a single mutation alone does not produce, say, an epilepsy phenotype, there would be provided one or more additional isolated mammalian polypeptides having biological functions as part of an ion channel in a mammal, wherein a mutation event selected from the group consisting of substitutions, deletions, truncations, insertions and rearrangements has occurred so as to affect the functioning of the ion channel. The cumulative effect of the mutations in each isolated mammalian polypeptide *in vivo* being to produce epilepsy or another neuro/physiological disorder in said mammal. The mutations may be in polypeptide subunits belonging to the same ion channel as described above, but may also be in polypeptide subunits that belong to different ion channels.

Typically the mutation is an amino acid substitution and the ion channel is a voltage-gated channel such as a sodium, potassium, calcium or chloride channel or a ligand-gated channel such as a member of the nAChR/GABA super family of receptors, or a functional fragment or homologue thereof.

Mutation combinations may be selected from, but are not restricted to, those represented in Table 1.

Accordingly, in a further aspect of the present invention there is provided an isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit wherein a mutation event selected from the group  
 5 consisting of the mutation events set forth in the following Table:

Subunit Gene	Amino Acid Change
SCN1A	R222X
SCN1A	W384X
SCN1A	A395P
SCN1A	F403L
SCN1A	Y413N
SCN1A	V422E
SCN1A	R1407X
SCN1A	M1780T
SCN1A	R1892X
SCN1B	R85H
SCN2A	R223Q
SCN2A	V892I
SCN2A	L1003I
SCN2A	T1200A
SCN2A	R1319Q
CHRNA5	V134I
CHRNA2	A125T
CHRNA3	R37H
KCNQ2	K69fsX119
KCNQ2	M1V
KCNQ2	M1T
KCNQ2	R353G
KCNQ2	R430X
KCNQ2	R570S
KCNQ2	L619R

has occurred.

In a further aspect of the invention there is provided an isolated polypeptide, said polypeptide being a  
 10 mutant or variant ion channel subunit wherein a mutation event has occurred such that the polypeptide has the amino acid sequence set forth in one of SEQ ID Numbers: 73-95. The mutation event disrupts the functioning of an ion channel so as to produce a phenotype of epilepsy, and/or  
 15 one or more other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic

ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperreflexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness.

In a particularly preferred embodiment of the present invention, the isolated polypeptide has an amino acid sequence as shown in any one of SEQ ID Numbers: 73-95. The sequences correspond to the novel amino acid changes laid out in Table 1 for those instances where the DNA mutation results in an amino acid change.

According to still another aspect of the present invention there is provided an isolated polypeptide, said polypeptide being a mutant KCNQ2 subunit, wherein the mutation event has occurred in the C-terminal domain of the KCNQ2 subunit and leads to a disturbance in the calmodulin binding affinity of the subunit, so as to produce an epilepsy phenotype.

In one form of the invention the mutations are substitutions in which an arginine residue is replaced with a glycine residue, or a leucine residue is replaced with an arginine. Preferably the substitutions are R353G and L619R transitions as illustrated by SEQ ID NOS: 92 and 95 respectively.

In a further form of the invention the mutations result in the replacement of an arginine for a stop codon, or an arginine is replaced with a serine. Preferably the mutations are R430X and R570S transitions as illustrated by SEQ ID NOS: 93 and 94 respectively.

In a still further aspect of the present invention there is provided a combination of two or more isolated polypeptides each having a novel mutation event as laid out in Table 1. The cumulative effect of the mutations in each isolated polypeptide molecule *in vivo* is to produce

an epilepsy or another disorder associated with ion channel dysfunction as described above in said mammal.

In a particularly preferred embodiment of the present invention, the isolated polypeptides have an amino acid  
5 sequence as shown in any one of SEQ ID Numbers: 73-95. The sequences correspond to the novel amino acid changes laid out in Table 1.

According to still another aspect of the present invention there is provided an isolated polypeptide  
10 comprising the amino acid sequence set forth in any one of SEQ ID Numbers: 73-95.

According to still another aspect of the present invention there is provided a polypeptide consisting of the amino acid sequence set forth in any one of SEQ ID  
15 Numbers: 73-95.

According to still another aspect of the present invention there is provided a method of preparing a polypeptide, comprising the steps of:

- (1) culturing host cells transfected with an  
20 expression vector comprising a nucleic acid molecule as described above under conditions effective for polypeptide production; and
- (2) harvesting the mutant ion channel subunit.

The mutant ion channel subunit may be allowed to  
25 assemble with other subunits constituting the channel that are either wild-type or themselves mutant subunits, whereby the assembled ion channel is harvested.

According to still another aspect of the invention there is provided a polypeptide which is the product of  
30 the process described above.

Substantially purified protein or fragments thereof can then be used in further biochemical analyses to establish secondary and tertiary structure. Such methodology is known in the art and includes, but is not  
35 restricted to, X-ray crystallography of crystals of the proteins or of the assembled ion channel incorporating the proteins or by nuclear magnetic resonance (NMR).

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Determination of structure allows for the rational design of pharmaceuticals to interact with the ion channel as a whole or through interaction with a specific subunit protein (see drug screening below), alter the overall ion  
5 channel protein charge configuration or charge interaction with other proteins, or to alter its function in the cell.

It will be appreciated that the mutant ion channel subunits included as part of the present invention will be useful in further applications which include a variety of  
10 hybridisation and immunological assays to screen for and detect the presence of either a normal or mutated gene or gene product. The invention enables therapeutic methods for the treatment of epilepsy as well as other disorders associated with ion channel dysfunction and also enables  
15 methods for the diagnosis or prognosis of epilepsy as well as other disorders associated with ion channel dysfunction.

#### Therapeutic Applications

20 According to still another aspect of the invention there is provided a method of treating epilepsy as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia,  
25 myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney  
30 disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, comprising administering a selective antagonist, agonist or modulator of an ion channel or ion channel subunit, when the ion  
35 channel contains a mutation in a subunit comprising the channel, as described above, to a subject in need of such treatment. Said mutation event may be causative of the



disorder when expressed alone or when expressed in combination with one or more additional mutations in subunits of the same or different ion channels, which are typically those identified in Table 1.

5 In still another aspect of the invention there is provided the use of a selective antagonist, agonist or modulator of an ion channel or ion channel subunit when the ion channel contains a mutation in a subunit comprising the channel, as described above, said mutation  
10 being causative of epilepsy as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's  
15 disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of  
20 infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, when expressed alone or when expressed in combination with a second mutation in a subunit of the same or different ion channel, as described above, in the manufacture of a medicament for  
25 the treatment of the disorder.

In one aspect, a suitable antagonist, agonist or modulator will restore wild-type function to the ion channel or channels containing the mutations of the present invention, or will negate the effects the mutant  
30 channel or channels have on cell function.

Using methods well known in the art, a mutant ion channel may be used to produce antibodies specific for the mutant channel that is causative of the disease or to screen libraries of pharmaceutical agents to identify  
35 those that bind the mutant ion channel.

In one aspect, an antibody, which specifically binds to a mutant ion channel or mutant ion channel subunit of

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the invention, may be used directly as an agonist, antagonist or modulator, or indirectly as a targeting or delivery mechanism for bringing a pharmaceutical agent to cells or tissues that express the mutant ion channel.

5 In a still further aspect of the invention there is provided an antibody which is immunologically reactive with a polypeptide as described above, but not with a wild-type ion channel or ion channel subunit thereof.

10 In particular, there is provided an antibody to an assembled ion channel containing a mutation in a subunit comprising the channel, which is causative of epilepsy or another disorder associated with ion channel dysfunction when expressed alone or when expressed in combination with one or more other mutations in subunits of the same or  
15 different ion channels. Such antibodies may include, but are not limited to, polyclonal, monoclonal, chimeric, and single chain antibodies as would be understood by the person skilled in the art.

For the production of antibodies, various hosts  
20 including rabbits, rats, goats, mice, humans, and others may be immunized by injection with a polypeptide as described above or with any fragment or oligopeptide thereof which has immunogenic properties. Various adjuvants may be used to increase immunological response  
25 and include, but are not limited to, Freund's, mineral gels such as aluminium hydroxide, and surface-active substances such as lysolecithin. Adjuvants used in humans include BCG (bacilli Calmette-Guerin) and Corynebacterium parvum.

30 It is preferred that the oligopeptides, peptides, or fragments used to induce antibodies to the mutant ion channel have an amino acid sequence consisting of at least 5 amino acids, and, more preferably, of at least 10 amino acids. It is also preferable that these oligopeptides,  
35 peptides, or fragments are identical to a portion of the amino acid sequence of the natural protein and contain the entire amino acid sequence of a small, naturally occurring

molecule. Short stretches of ion channel amino acids may be fused with those of another protein, such as K<sub>L</sub>H, and antibodies to the chimeric molecule may be produced.

Monoclonal antibodies to a mutant ion channel may be prepared using any technique which provides for the production of antibody molecules by continuous cell lines in culture. These include, but are not limited to, the hybridoma technique, the human B-cell hybridoma technique, and the EBV-hybridoma technique. (For example, see Kohler et al., 1975; Kozbor et al., 1985; Cote et al., 1983; Cole et al., 1984).

Monoclonal antibodies produced may include, but are not limited to, mouse-derived antibodies, humanised antibodies and fully human antibodies.

Antibodies may also be produced by inducing *in vivo* production in the lymphocyte population or by screening immunoglobulin libraries or panels of highly specific binding reagents as disclosed in the literature. (For example, see Orlandi et al., 1989; Winter and Milstein, 1991).

Antibody fragments which contain specific binding sites for a mutant ion channel may also be generated. For example, such fragments include, F(ab')<sub>2</sub> fragments produced by pepsin digestion of the antibody molecule and Fab fragments generated by reducing the disulfide bridges of the F(ab')<sub>2</sub> fragments. Alternatively, Fab expression libraries may be constructed to allow rapid and easy identification of monoclonal Fab fragments with the desired specificity. (For example, see Huse et al., 1989).

Various immunoassays may be used for screening to identify antibodies having the desired specificity. Numerous protocols for competitive binding or immunoradiometric assays using either polyclonal or monoclonal antibodies with established specificities are well known in the art. Such immunoassays typically involve the measurement of complex formation between an ion channel and its specific antibody. A two-site, monoclonal-

based immunoassay utilizing antibodies reactive to two non-interfering ion channel epitopes is preferred, but a competitive binding assay may also be employed.

In a further aspect of the invention there is provided a method of treating epilepsy as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, comprising administering an isolated nucleic acid molecule which is the complement (antisense) of any one of the nucleic acid molecules described above and which encodes an RNA molecule that hybridizes with the mRNA encoding a mutant ion channel subunit of the invention, to a subject in need of such treatment.

In a still further aspect of the invention there is provided the use of an isolated nucleic acid molecule which is the complement (antisense) of a nucleic acid molecule of the invention and which encodes an RNA molecule that hybridizes with the mRNA encoding a mutant ion channel subunit of the invention, in the manufacture of a medicament for the treatment of epilepsy as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney

disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

Typically, a vector expressing the complement  
5 (antisense) of the polynucleotides of the invention may be administered to a subject in need of such treatment. Many methods for introducing vectors into cells or tissues are available and equally suitable for use *in vivo*, *in vitro*, and *ex vivo*. For *ex vivo* therapy, vectors may be  
10 introduced into stem cells taken from the patient and clonally propagated for autologous transplant back into that same patient. Delivery by transfection, by liposome injections, or by polycationic amino polymers may be achieved using methods which are well known in the art.  
15 (For example, see Goldman et al., 1997).

Additional antisense or gene-targeted silencing strategies may include, but are not limited to, the use of antisense oligonucleotides, injection of antisense RNA, transfection of antisense RNA expression vectors, and the  
20 use of RNA interference (RNAi) or short interfering RNAs (siRNA). Still further, catalytic nucleic acid molecules such as DNazymes and ribozymes may be used for gene silencing (Breaker and Joyce, 1994; Haseloff and Gerlach, 1988). These molecules function by cleaving their target  
25 mRNA molecule rather than merely binding to it as in traditional antisense approaches.

In a further aspect, a suitable agonist, antagonist or modulator may include peptides, phosphopeptides or small organic or inorganic compounds that can restore  
30 wild-type activity of ion channels containing mutations in the subunits which comprise the channels as described above.

Peptides, phosphopeptides or small organic or inorganic compounds suitable for therapeutic applications  
35 may be identified using nucleic acids and peptides of the invention in drug screening applications as described below. Molecules identified from these screens may also be

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of therapeutic application in affected individuals carrying other ion channel subunit gene mutations if the molecule is able to correct the common underlying functional deficit imposed by these mutations and those of  
5 the invention.

There is therefore provided a method of treating epilepsy as well as other disorders associated with ion channel dysfunction comprising administering a compound that is a suitable agonist, antagonist or modulator of an  
10 ion channel and that has been identified using the mutant ion channel subunits of the invention.

In some instances, an appropriate approach for treatment may be combination therapy. This may involve the administering an antibody or complement (antisense) to a  
15 mutant ion channel or ion channel subunit of the invention to inhibit its functional effect, combined with administration of wild-type ion channel subunits which may restore levels of wild-type ion channel formation to normal levels. Wild-type ion channel subunits of the  
20 invention can be administered using gene therapy approaches as described above for complement administration.

There is therefore provided a method of treating epilepsy as well as other disorders associated with ion  
25 channel dysfunction comprising administration of an antibody or complement to a mutant ion channel or ion channel subunit of the invention in combination with administration of wild-type ion channel subunits.

In still another aspect of the invention there is  
30 provided the use of an antibody or complement to a mutant ion channel or ion channel subunit of the invention in combination with the use of wild-type ion channel subunits, in the manufacture of a medicament for the treatment of epilepsy as well as other disorders  
35 associated with ion channel dysfunction.

In further embodiments, any of the agonists, antagonists, modulators, antibodies, complementary

sequences or vectors of the invention may be administered in combination with other appropriate therapeutic agents. Selection of the appropriate agents may be made by those skilled in the art, according to conventional pharmaceutical principles. The combination of therapeutic agents may act synergistically to effect the treatment or prevention of the various disorders described above. Using this approach, therapeutic efficacy with lower dosages of each agent may be possible, thus reducing the potential for adverse side effects.

Any of the therapeutic methods described above may be applied to any subject in need of such therapy, including, for example, mammals such as dogs, cats, cows, horses, rabbits, monkeys, and most preferably, humans.

#### Drug Screening

According to still another aspect of the invention, nucleic acid molecules of the invention as well as peptides of the invention, particularly purified mutant ion channel subunit polypeptide and cells expressing these, are useful for the screening of candidate pharmaceutical agents for the treatment of epilepsy as well as other as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

Still further, it provides the use of a polypeptide complex for the screening of candidate pharmaceutical compounds.

Still further, it provides the use wherein high throughput screening techniques are employed.

Compounds that can be screened in accordance with the invention include, but are not limited to peptides (such as soluble peptides), phosphopeptides and small organic or inorganic molecules (such as natural product or synthetic chemical libraries and peptidomimetics).

In one embodiment, a screening assay may include a cell-based assay utilising eukaryotic or prokaryotic host cells that are stably transformed with recombinant molecules expressing the polypeptides or fragments of the invention, in competitive binding assays. Binding assays will measure the formation of complexes between a specific mutant ion channel subunit polypeptide or ion channel incorporating a mutant ion channel subunit polypeptide, and the compound being tested, or will measure the degree to which a compound being tested will inhibit or restore the formation of a complex between a specific mutant ion channel subunit polypeptide or ion channel incorporating a mutant ion channel subunit polypeptide, and its interactor or ligand.

The invention is particularly useful for screening compounds by using the polypeptides of the invention in transformed cells, transfected or injected oocytes, or animal models bearing mutated ion channel subunits such as transgenic animals or gene targeted (knock-in) animals (see transformed hosts). Drug candidates can be added to cultured cells that express a single mutant ion channel subunit or combination of mutant ion channel subunits (appropriate wild-type ion channel subunits should also be expressed for receptor assembly), can be added to oocytes transfected or injected with either a mutant ion channel subunit or combination of mutant ion channel subunits (appropriate wild-type ion channel subunits must also be injected for receptor assembly), or can be administered to an animal model containing a mutant ion channel or combination of mutant ion channels. Determining the



ability of the test compound to modulate mutant ion channel activity can be accomplished by a number of techniques known in the art. These include for example measuring the effect on the current of the channel (e.g. calcium-, chloride-, sodium-, potassium-ion flux) as compared to the current of a cell or animal containing wild-type ion channels. Current in cells can be measured by a number of approaches including the patch-clamp technique (methods described in Hamill et al, 1981) or using fluorescence based assays as are known in the art (see Gonzalez et al. 1999). Drug candidates that alter the current to a more normal level are useful for treating or preventing epilepsy as well as other disorders associated with ion channel dysfunction.

Non cell-based assays may also be used for identifying compounds that can inhibit or restore binding between the polypeptides of the invention or ion channels incorporating the polypeptides of the invention, and their interactors. Such assays are known in the art and include for example AlphaScreen technology (PerkinElmer Life Sciences, MA, USA). This application relies on the use of beads such that each interaction partner is bound to a separate bead via an antibody. Interaction of each partner will bring the beads into proximity, such that laser excitation initiates a number of chemical reactions ultimately leading to fluorophores emitting a light signal. Candidate compounds that inhibit the binding of the mutant ion channel subunit, or ion channel incorporating the mutant subunit, with its interactor will result in loss of light emission, while candidate compounds that restore the binding of the mutant ion channel subunit, or ion channel incorporating the mutant subunit, with its interactor will result in positive light emission. These assays ultimately enable identification and isolation of the candidate compounds.

High-throughput drug screening techniques may also employ methods as described in WO84/03564. Small peptide

test compounds synthesised on a solid substrate can be assayed for mutant ion channel subunit polypeptide or mutant ion channel binding. Bound mutant ion channel or mutant ion channel subunit polypeptide is then detected by methods well known in the art. In a variation of this technique, purified polypeptides of the invention can be coated directly onto plates to identify interacting test compounds.

The invention also contemplates the use of competition drug screening assays in which neutralizing antibodies capable of specifically binding the mutant ion channel compete with a test compound for binding thereto. In this manner, the antibodies can be used to detect the presence of any peptide that shares one or more antigenic determinants of the mutant ion channel.

The polypeptides of the present invention may also be used for screening compounds developed as a result of combinatorial library technology. This provides a way to test a large number of different substances for their ability to modulate activity of a polypeptide. A substance identified as a modulator of polypeptide function may be peptide or non-peptide in nature. Non-peptide "small molecules" are often preferred for many *in vivo* pharmaceutical applications. In addition, a mimic or mimetic of the substance may be designed for pharmaceutical use. The design of mimetics based on a known pharmaceutically active compound ("lead" compound) is a common approach to the development of novel pharmaceuticals. This is often desirable where the original active compound is difficult or expensive to synthesise or where it provides an unsuitable method of administration. In the design of a mimetic, particular parts of the original active compound that are important in determining the target property are identified. These parts or residues constituting the active region of the compound are known as its pharmacophore. Once found, the pharmacophore structure is modelled according to its

physical properties using data from a range of sources including x-ray diffraction data and NMR. A template molecule is then selected onto which chemical groups which mimic the pharmacophore can be added. The selection can be made such that the mimetic is easy to synthesise, is likely to be pharmacologically acceptable, does not degrade *in vivo* and retains the biological activity of the lead compound. Further optimisation or modification can be carried out to select one or more final mimetics useful for *in vivo* or clinical testing.

It is also possible to isolate a target-specific antibody and then solve its crystal structure. In principle, this approach yields a pharmacophore upon which subsequent drug design can be based as described above. It may be possible to avoid protein crystallography altogether by generating anti-idiotypic antibodies (anti-ids) to a functional, pharmacologically active antibody. As a mirror image of a mirror image, the binding site of the anti-ids would be expected to be an analogue of the original receptor. The anti-id could then be used to isolate peptides from chemically or biologically produced peptide banks.

Another alternative method for drug screening relies on structure-based rational drug design. Determination of the three dimensional structure of the polypeptides of the invention, or the three dimensional structure of the ion channels which incorporate these polypeptides allows for structure-based drug design to identify biologically active lead compounds.

Three dimensional structural models can be generated by a number of applications, some of which include experimental models such as x-ray crystallography and NMR and/or from *in silico* studies of structural databases such as the Protein Databank (PDB). In addition, three dimensional structural models can be determined using a number of known protein structure prediction techniques based on the primary sequences of the polypeptides (e.g.

SYBYL - Tripos Associated, St. Louis, MO), *de novo* protein structure design programs (e.g. MODELER - MSI Inc., San Diego, CA, or MOE - Chemical Computing Group, Montreal, Canada) or *ab initio* methods (e.g. see US Patent Numbers 5 5331573 and 5579250).

Once the three dimensional structure of a polypeptide or polypeptide complex has been determined, structure-based drug discovery techniques can be employed to design biologically-active compounds based on these three 10 dimensional structures. Such techniques are known in the art and include examples such as DOCK (University of California, San Francisco) or AUTODOCK (Scripps Research Institute, La Jolla, California). A computational docking protocol will identify the active site or sites that are 15 deemed important for protein activity based on a predicted protein model. Molecular databases, such as the Available Chemicals Directory (ACD) are then screened for molecules that complement the protein model.

Using methods such as these, potential clinical drug 20 candidates can be identified and computationally ranked in order to reduce the time and expense associated with typical 'wet lab' drug screening methodologies.

Compounds identified through screening procedures as described above, and which are based on the use of the 25 mutant nucleic acid and polypeptides of the invention, can also be tested for their effect on correcting the functional deficit imposed by other gene mutations in affected individuals including other ion channel subunit mutations.

30 Such compounds form a part of the present invention, as do pharmaceutical compositions containing these and a pharmaceutically acceptable carrier.

#### Pharmaceutical Preparations

35 Compounds identified from screening assays and shown to restore ion channel wild-type activity can be administered to a patient at a therapeutically effective

dose to treat or ameliorate epilepsy as well as other disorders associated with ion channel dysfunction, as described above. A therapeutically effective dose refers to that amount of the compound sufficient to result in  
5 amelioration of symptoms of the disorder.

Toxicity and therapeutic efficacy of such compounds can be determined by standard pharmaceutical procedures in cell cultures or experimental animals. The data obtained from these studies can then be used in the formulation of  
10 a range of dosages for use in humans.

Pharmaceutical compositions for use in accordance with the present invention can be formulated in a conventional manner using one or more physiological acceptable carriers, excipients or stabilisers which are  
15 well known. Acceptable carriers, excipients or stabilizers are non-toxic at the dosages and concentrations employed, and include buffers such as phosphate, citrate, and other organic acids; antioxidants including ascorbic acid; low molecular weight (less than about 10 residues)  
20 polypeptides; proteins, such as serum albumin, gelatin, or immunoglobulins; binding agents including hydrophilic polymers such as polyvinylpyrrolidone; amino acids such as glycine, glutamine, asparagine, arginine or lysine; monosaccharides, disaccharides, and other carbohydrates  
25 including glucose, mannose, or dextrans; chelating agents such as EDTA; sugar alcohols such as mannitol or sorbitol; salt-forming counterions such as sodium; and/or non-ionic surfactants such as Tween, Pluronic or polyethylene glycol (PEG).

30 The formulation of pharmaceutical compositions for use in accordance with the present invention will be based on the proposed route of administration. Routes of administration may include, but are not limited to, inhalation, insufflation (either through the mouth or  
35 nose), oral, buccal, rectal or parental administration.

### Diagnostic and Prognostic Applications

Polynucleotide sequences encoding an ion channel subunit may be used for the diagnosis or prognosis of epilepsy, as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, and the use of the nucleic acid molecules incorporated as part of the invention in diagnosis or prognosis of these disorders, or a predisposition to these disorders, is therefore contemplated. The nucleic acid molecules incorporating the novel mutation events laid out in Table 1 may be used for this purpose.

The polynucleotides that may be used for diagnostic or prognostic purposes include oligonucleotide sequences, genomic DNA and complementary RNA and DNA molecules. The polynucleotides may be used to detect and quantitate gene expression in biological samples. Genomic DNA used for the diagnosis or prognosis may be obtained from body cells, such as those present in the blood, tissue biopsy, surgical specimen, or autopsy material. The DNA may be isolated and used directly for detection of a specific sequence or may be amplified by the polymerase chain reaction (PCR) prior to analysis. Similarly, RNA or cDNA may also be used, with or without PCR amplification. To detect a specific nucleic acid sequence, hybridisation using specific oligonucleotides, restriction enzyme digest and mapping, PCR mapping, RNase protection, and various other methods may be employed. Oligonucleotides specific

to particular sequences can be chemically synthesized and labelled radioactively or nonradioactively and hybridised to individual samples immobilized on membranes or other solid-supports or in solution. The presence, absence or  
5 excess expression of any one of the mutant ion channel genes of the invention may then be visualized using methods such as autoradiography, fluorometry, or colorimetry.

In a further diagnostic or prognostic approach, the  
10 nucleotide sequences of the invention may be useful in assays that detect the presence of associated disorders, particularly those mentioned previously. The nucleotide sequences may be labelled by standard methods and added to a fluid or tissue sample from a patient under conditions  
15 suitable for the formation of hybridisation complexes. After a suitable incubation period, the sample is washed and the signal is quantitated and compared with a standard value. If the amount of signal in the patient sample is significantly altered in comparison to a control sample  
20 then the presence of altered levels of nucleotide sequences in the sample indicates the presence of the associated disorder. Such assays may also be used to evaluate the efficacy of a particular therapeutic treatment regimen in animal studies, in clinical trials,  
25 or to monitor the treatment of an individual patient.

In order to provide a basis for the diagnosis or prognosis of epilepsy and other disorders as described above, which are associated with the ion channel subunit mutations or variants of the invention, the nucleotide  
30 sequence of each gene can be compared between normal tissue and diseased tissue in order to establish whether the patient expresses a mutant gene.

In order to provide a basis for the diagnosis or prognosis of a disorder associated with abnormal  
35 expression of an ion channel subunit gene of the invention, a normal or standard profile for expression is established. This may be accomplished by combining body

fluids or cell extracts taken from normal subjects, either animal or human, with a sequence, or a fragment thereof, encoding the relevant ion channel subunit gene, under conditions suitable for hybridisation or amplification.

5 Standard hybridisation may be quantified by comparing the values obtained from normal subjects with values from an experiment in which a known amount of a substantially purified polynucleotide is used. Another method to identify a normal or standard profile for expression of an  
10 ion channel subunit gene is through quantitative RT-PCR studies. RNA isolated from body cells of a normal individual is reverse transcribed and real-time PCR using oligonucleotides specific for the relevant gene is conducted to establish a normal level of expression of the  
15 gene. Standard values obtained in both these examples may be compared with values obtained from samples from patients who are symptomatic for a disorder. Deviation from standard values is used to establish the presence of a disorder.

20 Once the presence of a disorder is established and a treatment protocol is initiated, hybridisation assays or quantitative RT-PCR studies may be repeated on a regular basis to determine if the level of expression in the patient begins to approximate that which is observed in  
25 the normal subject. The results obtained from successive assays may be used to show the efficacy of treatment over a period ranging from several days to months.

According to a further aspect of the invention there is provided the use of a polypeptide as described above in  
30 the diagnosis or prognosis of epilepsy as well as other disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia,  
35 migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain,



chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

5       When a diagnostic or prognostic assay is to be based upon proteins constituting an ion channel, a variety of approaches are possible. For example, diagnosis or prognosis can be achieved by monitoring differences in the electrophoretic mobility of normal and mutant proteins  
10       that form the ion channel. Such an approach will be particularly useful in identifying mutants in which charge substitutions are present, or in which insertions, deletions or substitutions have resulted in a significant change in the electrophoretic migration of the resultant  
15       protein. Alternatively, diagnosis or prognosis may be based upon differences in the proteolytic cleavage patterns of normal and mutant proteins, differences in molar ratios of the various amino acid residues, or by functional assays demonstrating altered function of the  
20       gene products.

      In another aspect, antibodies that specifically bind mutant ion channels may be used for the diagnosis or prognosis of a disorder, or in assays to monitor patients being treated with a complete ion channel or agonists,  
25       antagonists, modulators or inhibitors of an ion channel. Antibodies useful for diagnostic or prognostic purposes may be prepared in the same manner as described above for therapeutics. Diagnostic or prognostic assays for ion  
30       channels include methods that utilize the antibody and a label to detect a mutant ion channel in human body fluids or in extracts of cells or tissues. The antibodies may be used with or without modification, and may be labelled by covalent or non-covalent attachment of a reporter molecule.

35       A variety of protocols for measuring the presence of mutant ion channels, including but not restricted to, ELISAs, RIAs, and FACS, are known in the art and provide a

basis for diagnosing or prognosing a disorder. The expression of a mutant ion channel or combination of mutant ion channels is established by combining body fluids or cell extracts taken from test mammalian subjects, preferably human, with antibody to the ion channel or channels under conditions suitable for complex formation. The amount of complex formation may be quantitated by various methods, preferably by photometric means. Antibodies specific for the mutant ion channels will only bind to individuals expressing the said mutant ion channels and not to individuals expressing only wild-type channels (ie normal individuals). This establishes the basis for diagnosing the disorder.

Once an individual has been diagnosed or prognosed with a disorder, effective treatments can be initiated as described above. Treatments can be directed to amend the combination of ion channel subunit mutations or may be directed to one mutation.

#### Microarray

In further embodiments, complete cDNAs, oligonucleotides or longer fragments derived from any of the polynucleotide sequences described herein may be used as probes in a microarray. The microarray can be used to diagnose or prognose epilepsy, as well as other disorders associated with ion channel dysfunction, through the identification of genetic variants, mutations, and polymorphisms in the ion channel subunits that form part of the invention, to understand the genetic basis of a disorder, or can be used to develop and monitor the activities of therapeutic agents.

According to a further aspect of the present invention, tissue material obtained from genetically modified non-human animal models generated as a result of the identification of specific ion channel subunit human mutations (see below), particularly those disclosed in the present invention, can be used in microarray experiments.

These experiments can be conducted to identify the level of expression of specific ion channel subunits, or the level of expression of any cDNA clone from whole-tissue libraries, in diseased tissue as opposed to normal control tissue. Variations in the expression level of genes, including ion channel subunits, between the two tissues indicates their possible involvement in the disease process either as a cause or consequence of the original ion channel subunit mutation present in the animal model. These experiments may be used to determine gene function, to understand the genetic basis of a disorder, to diagnose or prognose a disorder, and to develop and monitor the activities of therapeutic agents. Microarrays may be prepared, used, and analyzed using methods known in the art. (For example, see Schena et al., 1996; Heller et al., 1997).

#### Transformed Hosts

The present invention also provides for the production of genetically modified (knock-out, knock-in and transgenic), non-human animal models comprising nucleic acid molecules containing the novel ion channel mutations or variants as laid out in Table 1. These animals are useful for the study of the function of ion channels, to study the mechanisms by which combinations of mutations in ion channel subunits interact to give rise to disease and the effects of these mutations on tissue development, for the screening of candidate pharmaceutical compounds, for the creation of explanted mammalian cell cultures which express mutant ion channels or combinations of mutant ion channels, and for the evaluation of potential therapeutic interventions.

Animal species which are suitable for use in the animal models of the present invention include, but are not limited to, rats, mice, hamsters, guinea pigs, rabbits, dogs, cats, goats, sheep, pigs, and non-human primates such as monkeys and chimpanzees. For initial

studies, genetically modified mice and rats are highly desirable due to the relative ease in generating knock-in, knock-out or transgenics of these animals, their ease of maintenance and their shorter life spans. For certain studies, transgenic yeast or invertebrates may be suitable and preferred because they allow for rapid screening and provide for much easier handling. For longer term studies, non-human primates may be desired due to their similarity with humans.

To create an animal model for a mutated ion channel, or an animal model incorporating a combination of mutations, several methods can be employed. These include, but are not limited to, generation of a specific mutation in a homologous animal gene, insertion of a wild type human gene and/or a humanized animal gene by homologous recombination, insertion of a mutant (single or multiple) human gene as genomic or minigene cDNA constructs using wild type or mutant or artificial promoter elements, or insertion of artificially modified fragments of the endogenous gene by homologous recombination. The modifications include insertion of mutant stop codons, the deletion of DNA sequences, or the inclusion of recombination elements (lox p sites) recognized by enzymes such as Cre recombinase.

To create transgenic mice in order to study gain of gene function *in vivo*, any mutant ion channel subunit gene of the invention can be inserted into a mouse germ line using standard techniques such as oocyte microinjection. Gain of gene function can mean the over-expression of a gene and its protein product, or the genetic complementation of a mutation of the gene under investigation. For oocyte injection, one or more copies of the mutant gene can be inserted into the pronucleus of a just-fertilized mouse oocyte. This oocyte is then reimplanted into a pseudo-pregnant foster mother. The live-born mice can then be screened for integrants using analysis of tail DNA for the presence of the relevant

human ion channel subunit gene sequence. The transgene can be either a complete genomic sequence injected as a YAC, BAC, PAC or other chromosome DNA fragment, a cDNA with either the natural promoter or a heterologous promoter, or a minigene containing all of the coding region and other elements found to be necessary for optimum expression.

To generate knock-out mice or knock-in mice, gene targeting through homologous recombination in mouse embryonic stem (ES) cells may be applied. Knock-out mice are generated to study loss of gene function *in vivo* while knock-in mice (which are preferred) allow the study of gain of function or to study the effect of specific gene mutations. Knock-in mice are similar to transgenic mice however the integration site and copy number are defined in the former.

For knock-out mouse generation, gene targeting vectors can be designed such that they delete (knock-out) the protein coding sequence of the relevant ion channel subunit gene in the mouse genome. In contrast, knock-in mice can be produced whereby a gene targeting vector containing the relevant ion channel subunit gene can integrate into a defined genetic locus in the mouse genome. For both applications, homologous recombination is catalysed by specific DNA repair enzymes that recognise homologous DNA sequences and exchange them via double crossover.

Gene targeting vectors are usually introduced into ES cells using electroporation. ES cell integrants are then isolated via an antibiotic resistance gene present on the targeting vector and are subsequently genotyped to identify those ES cell clones in which the gene under investigation has integrated into the locus of interest. The appropriate ES cells are then transmitted through the germline to produce a novel mouse strain.

In instances where gene ablation results in early embryonic lethality, conditional gene targeting may be employed. This allows genes to be deleted in a temporally

and spatially controlled fashion. As above, appropriate ES cells are transmitted through the germline to produce a novel mouse strain, however the actual deletion of the gene is performed in the adult mouse in a tissue specific or time controlled manner. Conditional gene targeting is most commonly achieved by use of the cre/lox system. The enzyme cre is able to recognise the 34 base pair loxP sequence such that loxP flanked (or floxed) DNA is recognised and excised by cre. Tissue specific cre expression in transgenic mice enables the generation of tissue specific knock-out mice by mating gene targeted floxed mice with cre transgenic mice. Knock-out can be conducted in every tissue (Schwenk et al., 1995) using the 'deleter' mouse or using transgenic mice with an inducible cre gene (such as those with tetracycline inducible cre genes), or knock-out can be tissue specific for example through the use of the CD19-cre mouse (Rickert et al., 1997).

Once knock-in animals have been produced which contain a specific mutation in a particular ion channel subunit, mating combinations may be initiated between such animals so as to produce progeny containing combinations of two or more ion channel mutations. These animals effectively mimic combinations of mutations that are proposed to cause human IGE cases. These animal models can subsequently be used to study the extent and mechanisms of disease as related to the mutated ion channel combinations, as well as for the screening of candidate therapeutic compounds.

According to still another aspect of the invention there is provided the use of genetically modified non-human animals as described above for the screening of candidate pharmaceutical compounds (see drug screening above). These animals are also useful for the evaluation (eg therapeutic efficacy, toxicity, metabolism) of candidate pharmaceutical compounds, including those identified from the invention as described above, for the

treatment of epilepsy as well as other as other disorders associated with ion channel dysfunction as described above.

5 It will be clearly understood that, although a number of prior art publications are referred to herein, this reference does not constitute an admission that any of these documents forms part of the common general knowledge in the art, in Australia or in any other country.

10 Throughout this specification and the claims, the words "comprise", "comprises" and "comprising" are used in a non-exclusive sense, except where the context requires otherwise.

15 It will be apparent to the person skilled in the art that while the invention has been described in some detail for the purposes of clarity and understanding, various modifications and alterations to the embodiments and methods described herein may be made without departing from the scope of the inventive concept disclosed in this specification.

20

#### Brief Description of the Drawings

Preferred forms of the invention will now be described, by way of example only, with reference to the following examples and the accompanying drawings, in  
25 which:

Figure 1 provides an example of ion channel subunit stoichiometry and the effect of multiple versus single ion channel subunit mutations. Figure 1A: A typical channel may have five subunits of three different types. Figure  
30 1B: In outbred populations complex diseases such as idiopathic generalized epilepsies may be due to mutations in two (or more) different subunit genes. Because only one allele of each subunit gene is abnormal, half the expressed subunits will have the mutation. Figure 1C: In  
35 inbred populations, both alleles of a single subunit gene will be affected, so all expressed subunits will be mutated. Figure 1D: Autosomal dominant disorders can be

attributed to single ion channel subunit mutations that give rise to severe functional consequences.

Figure 2 represents the location of mutations identified in the KCNQ2 ion channel subunit constituting the potassium channel. M: Missense mutation; T: Truncation mutation; F: Frameshift mutation; S: Splice site mutation.

Figure 3 provides examples of epilepsy pedigrees where mutation profiles of ion channel subunits for individuals constituting the pedigree have begun to be determined. These examples have been used to illustrate how the identification of novel ion channel subunit mutations and variations in IGE individuals can combine to give rise to the disorder.

Figure 4 shows the results of yeast two-hybrid analysis of R353G and L619R KCNQ2 mutants. Yeast were transformed with the empty DB (BAIT) plasmid (DBLeu), DB-Q2C wt, DB-Q2C R353G mutant or the DB-Q2 L619R mutant as indicated in A and the AD-CaM (TARGET) vector was introduced by gap-repair. Yeast control strains (Invitrogen™) were included on all plates for comparison. Control 1 has no interaction. Control 2 has a weak interaction. Control 3 has a moderately strong interaction. Control 4 has a strong interaction and control 5 has a very strong interaction. B. Growth of transformed yeast and controls on -leu -tryp selection. Yeast can grow on -leu if they contain the DB plasmid, and -tryp if they have AD plasmid. C. Growth of transformed yeast and controls on -leu -tryp -his +40mM 3AT after 48hrs. Yeast can grow on -his+3AT if the *his* reporter gene is activated by interaction between the BAIT and TARGET plasmids. D-F. *LacZ* Filter assay for interaction between BAIT and TARGET plasmids, photos taken after 2hrs (D), 7hrs (E) and 24hrs (F). Activation of the  $\beta$ -galactosidase reporter gene by interaction of the BAIT and TARGET plasmids leads to the dark appearance of colonies.

Figure 5 shows the results of CaM affinity experiments with the R353G and L619R KCNQ2 mutants. The



chart below shows the values from the CPRG assay for  $\beta$ -galactosidase activity as a measure of KCNQ2C-CaM binding efficiency. The area of each bar in the chart equates to the CaM binding efficiency of the BAIT. Broken lines  
5 indicate statistical comparison by Student's *t* test \*  $P < 0.01$ , \*\*  $P < 0.001$ .

#### Modes for Performing the Invention

Potassium channels are the most diverse class of ion  
10 channel. The *C. elegans* genome encodes about 80 different potassium channel genes and there are probably more in mammals. About ten potassium channel genes are known to be mutated in human disease and include four members of the KCNQ gene sub-family of potassium channels. KCNQ proteins  
15 have six transmembrane domains, a single P-loop that forms the selectivity filter of the pore, a positively charged fourth transmembrane domain that probably acts as a voltage sensor, and intracellular amino and carboxy termini. The C-terminus is long and contains a conserved  
20 "A domain" followed by a short stretch thought to be involved in subunit assembly.

Four KCNQ subunits are thought to combine to form a functional potassium channel. All five known KCNQ proteins can form homomeric channels *in vitro* and the formation of  
25 heteromers appears to be restricted to certain combinations. For instance KCNQ2 and KCNQ3, which are predominantly expressed in the central nervous system, form a heteromultimeric channel that mediates the neuronal muscarinic-regulated current (M-current), also known as  
30 the M-channel (or M-type  $K^+$  channel). The M-current is a slowly activating, non-inactivating potassium conductance known to regulate neuronal excitability by determining the firing properties of neurons and their responsiveness to synaptic input (Wang et al., 1998). Because it is the only  
35 current active at voltages near the threshold for action potential initiation, the M-current has a major impact on neuronal excitability.

Sodium (the alpha subunit) and calcium channels are thought to have evolved from the potassium channel subunit, and they each consist of four domains covalently linked as the one molecule, each domain being equivalent to one of the subunits that associate to form the potassium channel. Each of the four domains of the sodium and calcium channels are comprised of six transmembrane segments.

Voltage-gated sodium channels are required to generate the electrical excitation in neurones, heart and skeletal muscle fibres, which express tissue specific isoforms. Sodium channels are heteromers of a pore forming alpha subunit and a modulatory beta-1 subunit, with an additional beta-2 subunit in neuronal channels. Ten genes encoding sodium channel alpha subunits and 3 genes encoding different beta subunits have so far been identified. The beta subunits of the sodium channels do not associate with the alpha subunits to form any part of the pore, they do however affect the way the alpha pore forming subunit functions.

As with sodium channels, calcium channels consist of a single pore forming alpha subunit, of which at least six types have been identified to date, and several accessory subunits including four beta, one gamma and one alpha2-delta gene. Many of these subunits also encode multiple splice variants adding to the diversity of receptor subunits of this family of ion channels.

The ion channels in the nAChR/GABA super family show a theoretical pentameric channel. Gamma-Aminobutyric acid (GABA) is the most abundant inhibitory neurotransmitter in the central nervous system. GABA-ergic inhibition is mediated by two major classes of receptors, type A (GABA-A) and type B (GABA-B). GABA-B receptors are members of the class of receptors coupled to G-proteins and mediate a variety of inhibitory effects via secondary messenger cascades. GABA-A receptors are ligand-gated chloride channels that mediate rapid inhibition.

The GABA-A channel has 16 separate, but related, genes encoding subunits. These are grouped on the basis of sequence identity into alpha, beta, gamma, delta, epsilon, theta and pi subunits. There are six alpha subunits ( $\alpha 1$ - $\alpha 6$ ), three beta subunits ( $\beta 1$ - $\beta 3$ ) and three gamma subunits ( $\gamma 1$ - $\gamma 3$ ). Each GABA-A receptor comprises five subunits which may, at least in theory, be selected from any of these subunits.

Neuronal nicotinic acetylcholine receptors (nAChRs) consist of heterologous pentamers comprising various combinations of alpha subunits or alpha and beta subunits ( $\alpha 2$ - $\alpha 9$ ;  $\beta 2$ - $\beta 4$ ). The alpha subunits are characterised by adjacent cysteine residues at amino acid positions 192 and 193, and the beta subunits by the lack of these cysteine residues. They are ligand-gated ion channels differentially expressed throughout the brain to form physiologically and pharmacologically distinct receptors hypothesised to mediate fast, excitatory transmission between neurons of the central nervous system or to modulate neurotransmission from their presynaptic position.

In chicken and rat, the predominant nAChR subtype is composed of alpha-4 and beta-2 subunits. The transmembrane 2 (M2) segments of the subunits are arranged as alpha helices and contribute to the walls of the neurotransmitter-gated ion channel. The alpha helices appear to be kinked and orientated in such a way that the side chains of the highly conserved M2-leucine residues project inwards when the channel is closed. ACh is thought to cause a conformational change by altering the association of the amino acid residues of M2. The opening of the channel seems to be due to rotations of the gate forming side chains of the amino acid residues; the conserved polar serines and threonines may form the critical gate in the open channel.

Example 1: Identification of mutations in ion channels

Previous studies by reference (Wallace et al., 1998; PCT/AU01/00581; Wallace et al., 2001b; Australian patent AU-B-56247/96; Steinlein et al., 1995; PCT/AU01/00541; 5 Phillips et al., 2001; PCT/AU01/00729; PCT/AU01/01648; PCT/AU02/00910; Wallace et al., 2001a, the disclosures of which are incorporated herein by reference) have identified mutations in a number of ion channel subunits associated with epilepsy. These include ion channel 10 subunits of voltage-gated (eg SCN1A, SCN1B, KCNQ2, KCNQ3) or ligand-gated (eg CHRNA4, CHRNB2, GABRG2, GABRD) types. To identify further mutations in ion channel genes, subunits which comprise the ion channels were screened for molecular defects in epilepsy patients.

15 Human genomic sequence available from the Human Genome Project was used to characterize the genomic organisation for each subunit gene. Each gene was subsequently screened for sequence changes using single strand conformation polymorphism (SSCP) analysis in a 20 large sample of epileptics with common sporadic IGE subtypes eg juvenile myoclonic epilepsy (JME), childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE) and epilepsy with generalized tonic-clonic seizures (TCS). Clinical observations can then be compared to the 25 molecular defects characterized in order to establish the combinations of mutant subunits involved in the various disease states, and therefore to provide validated drug targets for each of these disease states. This will provide a basis for novel drug treatments directed at the 30 genetic defects present in each patient.

The coding sequence for each of the ion channel subunits was aligned with human genomic sequence present in available databases at the National Centre for Biotechnology Information (NCBI). The BLASTN algorithm was 35 typically used for sequence alignment and resulted in the genomic organisation (intron-exon structure) of each gene being determined. Where genomic sequence for an ion

channel subunit was not available, BACs or PACs containing the relevant ion channel subunit were identified through screening of high density filters containing these clones and were subsequently sequenced.

5        Availability of entire genomic sequence for each ion channel subunit facilitated the design of intronic primers spanning each exon. These primers were used for both high throughput SSCP screening and direct DNA sequencing.

10    Example 2: Sample preparation for SSCP screening

      A large collection of individuals affected with epilepsy have undergone careful clinical phenotyping and additional data regarding their family history has been collated. Informed consent was obtained from each  
15 individual for blood collection and its use in subsequent experimental procedures. Clinical phenotypes incorporated classical IGE cases as well as GEFS+ and febrile seizure cases.

      DNA was extracted from collected blood using the  
20 QIAamp DNA Blood Maxi kit (Qiagen) according to manufacturers specifications or through procedures adapted from Wyman and White (1980). Stock DNA samples were kept at a concentration of 1 ug/ul.

      In preparation for SSCP analysis, samples to be  
25 screened were formatted into 96-well plates at a concentration of 30 ng/ul. These master plates were subsequently used to prepare exon specific PCR reactions in the 96-well format.

30    Example 3: Identification of sequence alterations in ion channel genes

      SSCP analysis of specific ion channel exons followed by sequencing of SSCP bandshifts was performed on  
35 individuals constituting the 96-well plates to identify sequence alterations.

      Primers used for SSCP were labelled at their 5' end with HEX and typical PCR reactions were performed in a

total volume of 10  $\mu$ l. All PCR reactions contained 67 mM Tris-HCl (pH 8.8); 16.5 mM  $(\text{NH}_4)_2\text{SO}_4$ ; 6.5  $\mu$ M EDTA; 1.5 mM  $\text{MgCl}_2$ ; 200  $\mu$ M each dNTP; 10% DMSO; 0.17 mg/ml BSA; 10 mM  $\beta$ -mercaptoethanol; 5  $\mu$ g/ml each primer and 100 U/ml *Taq* DNA polymerase. PCR reactions were typically performed using 10 cycles of 94°C for 30 seconds, 60°C for 30 seconds, and 72°C for 30 seconds followed by 25 cycles of 94°C for 30 seconds, 55°C for 30 seconds, and 72°C for 30 seconds. A final extension reaction for 10 minutes at 72°C followed.

Ten to twenty  $\mu$ l of loading dye comprising 50% (v/v) formamide, 12.5 mM EDTA and 0.02% (w/v) bromophenol blue were added to completed reactions which were subsequently run on non-denaturing 4% polyacrylamide gels with a cross-linking ratio of 35:1 (acrylamide:bis-acrylamide) and containing 2% glycerol. Gel thickness was 100 $\mu$ m, width 168mm and length 160mm. Gels were run at 1200 volts and approximately 20mA, at 18°C and analysed on the GelScan 2000 system (Corbett Research, Australia) according to manufacturers specifications.

PCR products showing a conformational change were subsequently sequenced. This first involved re-amplification of the amplicon from the relevant individual (primers used in this instance did not contain 5' HEX labels) followed by purification of the PCR amplified templates for sequencing using QiaQuick PCR preps (Qiagen) based on manufacturers procedures. The primers used to sequence the purified amplicons were identical to those used for the initial amplification step. For each sequencing reaction, 25 ng of primer and 100 ng of purified PCR template were used. The BigDye sequencing kit (ABI) was used for all sequencing reactions according to the manufacturers specifications. The products were run on an ABI 377 Sequencer and analysed using the EditView program.

Table 1 shows the novel sequence changes identified in the ion channel subunits screened.

#### Example 4: Digenic model examples

In some instances a single mutation in an ion channel alone is insufficient to give rise to an epilepsy phenotype. However combinations of mutations each  
5 conferring a subtle change of function to an ion channel, as proposed by the digenic model (PCT/AU01/00872), may be sufficient to produce an epilepsy phenotype.

Using mutations and variations in ion channel subunits previously identified, the digenic model may be  
10 validated through a parametric analysis of large families in which two abnormal alleles co-segregate by chance to identify mutations which act co-operatively to give an epilepsy phenotype. It is envisaged that the strategy of careful clinical phenotyping in these large families,  
15 together with a linkage analysis based on the digenic hypothesis will allow identification of the mutations in ion channels associated with IGEs. If molecular genetic studies in IGE are successful using the digenic hypothesis, such an approach might serve as a model for  
20 other disorders with complex inheritance.

The digenic hypothesis predicts that the closer the genetic relationship between affected individuals, the more similar the sub-syndromes, consistent with published data (Italian League Against Epilepsy Genetic  
25 Collaborative Group, 1993). This is because more distant relatives are less likely to share the same combinations of mutated subunits.

Identical twins have the same pair of mutated subunits and the same minor alleles so the sub-syndromes  
30 are identical. Affected sib-pairs, including dizygous twins, with the same sub-syndrome would also have the same pair of mutated subunits, but differences in minor alleles would lead to less similarity than with monozygous twins. Some sib-pairs and dizygous twins, have quite different  
35 sub-syndromes; this would be due to different combinations of mutated subunits, when the parents have more than two mutated alleles between them.

A special situation exists in inbred communities that parallels observations on autosomal recessive mouse models. Here the two mutated alleles of the digenic model are the same and thus result in a true autosomal recessive disorder. Because all affected individuals have the same pair of mutated alleles, and a similar genetic background, the phenotypes are very similar.

In outbred communities approximately 1% of the population would have IGE genotypes (2 mutated alleles) and 0.3% would clinically express IGE. Most of these would have mutations in two different channel subunits. In such communities most cases would appear "sporadic" as the risk to first degree relatives would be less than 10%.

For example, let there be three IGE loci (A,B,C) and let the frequency of abnormal alleles ( $a^*$ ,  $b^*$ ,  $c^*$ ) at each locus be .027 and of normal alleles ( $a$ ,  $b$ ,  $c$ ) be .973. Then, the distribution of genotypes  $aa^*$ ,  $a^*a$ ,  $a^*a^*$  and  $aa$  at locus A will be .0263 ( $.027 \times .973$ ), .0263, .0007 and .9467 respectively, and similarly for loci B and C. In this population .8485 will have no mutated alleles ( $.9467^3$ ), .1413 will have one mutated allele ( $a^*$  or  $b^*$  or  $c^*$ ;  $.0263 \times .9467^2 \times 6$ ), .0098 will have two abnormal alleles (.0020 two same abnormal alleles, .0078, two different abnormal alleles) and 0.00037 will have more than two abnormal alleles. Thus in this population .01, or 1%, will have two or more abnormal alleles (IGE genotype), and the total abnormal allele frequency will be .08 ( $3 \times .027$ ).

To determine the familial risks and allele patterns in affected pairs, the frequency distribution of population matings and the percentage of children with 2 or more abnormal alleles must be determined. The frequency of matings with no abnormal alleles ( $0 \times 0$ ) is .72 ( $.8485^2$ ), for  $1 \times 0$  and  $0 \times 1$  matings .24 ( $2 \times .8485 \times .1413$ ), for a  $1 \times 1$  mating .020, and for  $2 \times 0$  and  $0 \times 2$  matings .0166 etc. From this distribution of matings the frequency of children with 2 or more abnormal alleles can



be shown to be .01. For example, the 0 x 2 and 2 x 0 matings contribute .0033 of this .01 frequency (.0166 [mating frequency] x .2 [chance of that mating producing a child with 2 or more abnormal alleles]).

5 To determine parental risk it can be shown that of children with 2 abnormal alleles (IGE genotype), .49 derive from 1 x 1 matings where no parent is affected, .33 derive from a 2 x 0 and 0 x 2 matings etc. For the 2 x 0 and 0 x 2 matings, half the parents have IGE genotypes and  
10 contribute .16 (.33/2) to the parental risk with the total parental risk of an IGE genotype being .258. The other matings that contribute to affected parent-child pairs are 2 x 1, 1 x 2, 3 x 0, 0 x 3 etc.

The sibling risk of an IGE genotype is .305. For  
15 example 2 x 0 and 0 x 2 matings contributed .08 to the sibling risk (.33[fraction of children with 2 abnormal alleles] x .25[the chance of that mating producing a child with 2 or more abnormal alleles]). Similarly the offspring risk was determined to be .248 by mating individuals with  
20 2 abnormal alleles with the general population. Thus at 30% penetrance the risk for IGE phenotype for parents of a proband is .077, for siblings .091, and for offspring .074.

It can be shown that affected sib pairs share the  
25 same abnormal allele pair in 85% of cases. This is because of all affected sib pairs 44% derive from 1 x 1 matings and 23% from 0 x 2 and 2 x 0 matings where all affected siblings have the same genotype. In contrast, 24% derive from 1 x 2 matings and 9% from 3 x 1 and 2 x 2 matings etc  
30 where affected sibling genotypes sometimes differ.

For affected parent-child pairs, genotypes are identical in only 58%. Of affected parent child pairs, 43% derive from 0 x 2 matings where genotypes are identical, whereas 38% derive from 0 x 3 and 17% from 1 x 2 where the  
35 majority of crosses yield different affected genotypes.

Based on the digenic model it has been postulated that most classical IGE and GEFS<sup>+</sup> cases are due to the

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combination of two mutations in multi-subunit ion channels. These are typically point mutations resulting in a subtle change of function. The critical postulate is that two mutations, usually, but not exclusively, in different subunit alleles ("digenic model"), are required for clinical expression of IGE.

The hypothesis that similar phenotypes can be caused by the combination of mutations in two (or more) different subunits (outbred communities), or by the same mutation in two (or more) alleles of the same subunit (inbred communities), may seem implausible. However, applying the digenic hypothesis to the theoretical pentameric channel shown in Figure 1, in outbred communities IGE will be due to subunit combinations such as  $\alpha^*\alpha\beta^*\beta\Delta$ ,  $\alpha^*\alpha\beta\beta\Delta^*$  or  $\alpha\alpha\beta^*\beta\Delta^*$  (mutated subunits indicated by \*). In inbred communities  $\alpha^*\alpha^*\beta\beta\Delta$  or  $\alpha\alpha\beta^*\beta^*\Delta$  combinations might cause IGE phenotypes. We assume that the mutations will not cause reduced expression of the alleles and that the altered ion channel excitability, and consequent IGE phenotype, caused by mutations in two different alleles is similar to that caused by the same mutation in both alleles of one subunit. Finally, subunit mutations with more severe functional consequences (eg breaking a disulphide bridge in SCN1B or amino acid substitution in the pore forming regions of SCN1A for GEFS<sup>+</sup>) cause autosomal dominant generalized epilepsies with a penetrance of 60-90%. Such "severe" mutations are rare (allele frequency <0.01%) and are infrequent causes of GEFS<sup>+</sup>. They very rarely, or perhaps never, cause classical IGE.

The relative separate segregation of classical IGE and GEFS<sup>+</sup> phenotypes is an anecdotal clinical observation of ours (Singh et al., 1999), although the separation is not absolute. The separation is supported by previous family and EEG studies of Doose and colleagues who described "type A" and "type B" liabilities which we may approximate the GEFS<sup>+</sup> and classical IGE groupings respectively (Doose and Baier, 1987).

The digenic model predicts that affected sib pairs will share the same genes in 85% of cases whereas they will have at least one different allele in the remaining 15%. In contrast, only 58% of parent-child pairs share the same alleles in a 3 locus model. Thus there should be greater similarity of syndromes between sibling pairs than parent-child pairs. This would be most objectively measured by age of onset and seizure types.

Estimates for the risk of febrile seizures or IGE in relatives vary. The estimates range from 5%-10% for siblings, 4%-6% for offspring, 3%-6% for parents, and 2-3% for grandparents. Underestimation may occur because IGE manifest in youth, and parents and particularly grandparents may be unaware of seizures in themselves in younger years. This is particularly true where there was stigma associated with epilepsy and where the epilepsy may have been mild and unrecognized. Underestimation of sibling and offspring risks occurs when unaffected young children are counted, some of whom will develop IGE in adolescence. Overestimation may occur with misdiagnosis of seizures or inclusion of seizures unrelated to IGE (e.g. due to trauma or tumors)

In autosomal dominant models the risk to affected relatives reduces proportionally (50% for first degree relatives, 25% for second degree etc). For all oligogenic or polygenic models the risk decreases more quickly. For a digenic model with three loci, the risks are 9.1% for siblings, 7.4% for offspring, 7.7% for parents. Rigorous measurement of the familial recurrence rates, with careful phenotyping and age-corrected risk estimates could be compared with the predictions from the digenic model, and it is proposed to do this.

There is a small amount of information on IGE families regarding haplotype distribution. For example, there is some evidence for a locus on 8q as determined by parametric linkage in a single family (Fong et al., 1998) and by non-parametric analysis in multiple small families

(Zara et al., 1995). Interestingly, in the latter study the 8q haplotype not infrequently came from the unaffected parent. This would be quite compatible with the digenic model and evaluation of other data sets in this manner could be used to test the hypothesis, and it is proposed to do this.

Following the analysis of one large family with epilepsy where the two main phenotypes were childhood absence epilepsy (CAE) and febrile seizures (FS), the inheritance of FS was found to be autosomal dominant and the penetrance 75%. However the inheritance of CAE in this family was not simple Mendelian, but suggestive of complex inheritance with the involvement of more than one gene. The power of this large family was used to explore the complex genetics of CAE further.

Linkage analysis on this family in which individuals with CAE, FS and FS+ were deemed affected led to the detection of linkage on chromosome 5q and identification of a mutation in the GABRG2 gene (R43Q) which is localised to this region (Wallace et al., 2001a; PCT/AU01/00729). All 10 tested individuals with FS alone in this family had this mutation and 7 CAE affected individuals in this family also had the mutation. To test the digenic model of IGEs in the CAE affected individuals, the whole genome screen of this family was reanalysed with only individuals with CAE considered affected. Linkage analysis was performed using FASTLINK v4.0, two-point lod scores were calculated assuming 50% penetrance and a 2% phenocopy rate and individuals with FS or FS+ were coded as unknown. Markers producing a lod score greater than 1 were reanalysed without a phenocopy rate and at the observed penetrance for CAE in this family (30%). Results from the analysis revealed significant linkage to chromosome 14q22-q23 (lod 3.4). This provides strong evidence for a second locus segregating with CAE affected individuals in this family. While the GABRG2 mutation is sufficient to cause FS, the CAE phenotype is thought to be due to both the

GABRG2 mutation and a mutation occurring in a gene mapping to the 14q locus, as proposed by the digenic model.

For the application of the digenic model to sporadic cases of IGE and affected individuals belonging to smaller families in which genotyping and linkage analysis is not a feasible approach to disease gene identification, direct mutation analysis of ion channel genes in these individuals has been carried out as described above. In Table 1 there is provided an indication of novel genetic alterations so far identified through mutation analysis screening of these individuals. Figure 2 provides an example to indicate where some of these mutations have occurred with respect to the potassium channel KCNQ2 gene.

The identification of novel mutations and variations in ion channel subunits in IGE individuals provides resources to further test the digenic hypothesis and mutation profiles are starting to accumulate for a number of subunit changes that are observed in the same individuals. Figure 3 provides results from some of these profiles.

Figure 3A shows a 3 generation family in which individual III-1 has myoclonic astatic epilepsy and contains a N43del mutation in the SCN3A gene as well as an A1067T mutation in the SCN1A gene. Individual I-1 also has the SCN3A mutation but alone this mutation is not sufficient to cause epilepsy in this individual. The SCN3A mutation has likely been inherited from the grandfather through the mother, while the SCN1A mutation is likely to arise from the father. Both parents are unaffected but have yet to be screened for the presence of the mutations in these subunits. Individual II-1 is likely to contain an as yet unidentified ion channel subunit mutation acting in co-operation with the SCN3A mutation already identified in this individual.

Figure 3B is another 3 generation family in which individual III-1 has myoclonic astatic epilepsy due to a combination of the same SCN3A and SCN1A mutations as

above. However, in this family both parents have febrile seizures most likely due to the presence of just one of the mutations in each parent, as proposed by the model. This is in contrast to individuals II-2 and II-3 in Figure 4A who also contain one of the mutations in these genes each. These individuals are phenotypically normal most likely due to incomplete penetrance of these mutations in each case.

Figure 3C shows a larger multi-generation family in which individual IV-5 has a mutation in both the SCN3A and GABRG2 subunits. In combination, these give rise to severe myoclonic epilepsy of infancy but alone either cause febrile seizures (GABRG2 mutation in III-3 and IV-4) or are without an effect (SCN3A mutation in III-2) as proposed by the model.

These examples therefore illustrate the digenic model as determined from mutation analysis studies of ion channel subunits in affected individuals and highlight the need to identify genetic alterations in the genes encoding ion channel subunits.

#### Example 5: Analysis of ion channels and ion channel subunits

The structure and function of the mutant ion channels and mutant ion channel subunits of the present invention can be determined using a variety of molecular biological studies. These studies may provide clues as to the mechanisms by which mutations in ion channel subunits effect the functioning of the ion channel. For instance the identification of proteins that interact with mutant ion channels (or whose interaction is impeded by a mutation in an ion channel subunit) may help determine the molecular mechanisms that are disrupted as a result of a mutation. Procedures such as the yeast two-hybrid system can be used to discover and identify such interacting proteins.

The principle behind the yeast two-hybrid procedure is that many eukaryotic transcriptional activators, including those in yeast, consist of two discrete modular domains. The first is a DNA-binding domain that binds to a specific promoter sequence and the second is an activation domain that directs the RNA polymerase II complex to transcribe the gene downstream of the DNA binding site. Both domains are required for transcriptional activation as neither domain can activate transcription on its own. In the yeast two-hybrid procedure, the gene of interest or parts thereof (BAIT), is cloned in such a way that it is expressed as a fusion to a peptide that has a DNA binding domain. A second gene, or number of genes, such as those from a cDNA library (TARGET), is cloned so that it is expressed as a fusion to an activation domain. Interaction of the protein of interest with its binding partner brings the DNA-binding peptide together with the activation domain and initiates transcription of the reporter genes. The first reporter gene will select for yeast cells that contain interacting proteins (this reporter is usually a nutritional gene required for growth on selective media). The second reporter is used for confirmation and while being expressed in response to interacting proteins it is usually not required for growth.

#### KCNQ2 interactors

Despite the identification of a number of KCNQ2 mutations responsible for epilepsy, including those of the present study, the underlying biological mechanisms responsible for the epilepsy remains largely uncharacterized. Towards identifying these mechanisms, the large intracellular C-terminal region of KCNQ2 was screened for interactions with other proteins using the yeast-two hybrid procedure. The C-terminus accounts for 63% of the KCNQ2 protein and, in common with other KCNQ subunits, contains a conserved 'A domain' (Jentsch, 2000; Schwake et al., 2000) thought to be involved in subunit

interactions as well as another distal short conserved region that has been associated with subunit assembly, at least in KCNQ1 (Jentsch, 2000; Schmitt et al., 2000).

5 A) Yeast-two hybrid analysis

A yeast two-hybrid screen was carried out using the ProQuest™ Two-Hybrid System with Gateway™ Technology (Invitrogen™) according to manufacturer's directions. A KCNQ2 C-terminal entry (BAIT) clone was generated using  
10 the pENTR Directional TOPO® Cloning Kit (Invitrogen™). The following primers were designed to amplify the intracellular C-terminal region of KCNQ2 based on the sequence of human KCNQ2 (Genbank accession number NM\_172107): KCNQ2F: 5'-CACCAAGGTTTCAGGAGCAGCACAGG-3' and  
15 KCNQ2R: 5'-TCACTTCCTGGGCCCGGCCAGCC-3'. The 1611 base pair cloned fragment included exon 10a (found in all our amplified clones), corresponding to amino acid 373-382 of the KCNQ2 protein. The extra 30 base pairs (10 amino acids) were included in our numbering. The PCR-product was  
20 cloned into the pENTR/D-TOPO® vector (Invitrogen™) via the TOPO® Cloning reaction according to the manufacturer's instructions. Following sequence verification, the KCNQ2 cDNA fragment was then subcloned into pDEST™32, the DNA Binding domain (DB) Gateway™ Destination Vector  
25 (Invitrogen™).

The ProQuest™ Two-Hybrid human brain cDNA Library (TARGET) with Gateway™ technology (ResGen™, Invitrogen™ Corporation) was amplified according to the manufacturer's instructions. Plasmid DNA was purified from the cell  
30 pellet using the HiSpeed Plasmid Maxi Kit (Qiagen) according to the manufacturer's instructions.

Both the DBLeu (empty bait vector) and DB-KCNQ2 wild-type (wt) C-term BAITs were transformed into the yeast strain Mav203 and plated onto minimal selective media  
35 lacking leucine. A duplicate was carried out where the empty library TARGET (pAD) vector was co-transformed in addition to each BAIT and plated onto minimal selective



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media lacking leucine (-leu) and tryptophan (-tryp). Yeast control strains (Invitrogen™) were included on all plates. Control 1, used as a negative control, contained empty plasmids pPC97 and pPC86. Control 2 had pPC97-RB and pPC86-E2F1, which express a relatively weak interaction. Control 3 contained plasmids encoding the *Drosophila* DP (pPC97) and E2F (pPC86) domains that have a moderately strong interaction, and provide a control for plasmid shuffling. Control 4 contained pPC97-Fos and pPC86-Jun which express a relatively strong interaction, and control 5 had a pCL1 plasmid encoding full-length GAL4p and empty pPC86 and was used as a positive control.

The constructs were tested for self-activation of the *his* and  $\beta$ -gal reporter genes according to Invitrogen™ instructions.

For the yeast-two hybrid screen, competent yeast cells were prepared for each BAIT (DB-KCNQ2 wt C-term construct) to be screened, transformed with 31µg of ProQuest™ Two-Hybrid human brain AD (activation domain)-cDNA Library and plated onto minimal selective media lacking leucine (-leu), tryptophan (-tryp) and histidine (-his) and containing 3-aminotriazole (+3AT). Positive colonies from each screen were PCR-amplified and re-introduced into fresh yeast cells containing the BAIT to re-test for two-hybrid interaction phenotypes. Those giving rise to more than one PCR product or that failed to re-test positively were systematically eliminated. Positives that re-tested were sequenced using the ABI PRISM® BigDye™ Terminators v3.0 technology. Once identified, the sequence of the potential interactor was checked to verify it was in the same translational frame as the Gal4p-AD encoding sequence of the prey construct.

Approximately  $3 \times 10^6$  clones from the ProQuest™ Two-Hybrid human brain cDNA Library were screened for interaction with the DB-Q2C wt bait. Among 1039 positive AD-cDNAs recovered, re-tested and subsequently sequenced

all were identified as the CALM2 gene, encoding the ubiquitous,  $\text{Ca}^{2+}$ -binding protein, Calmodulin (CaM).

The interaction between the C-terminal region of KCNQ2 and CaM has also been reported by other studies (Wen and Levitan, 2002; Yus-Najera et al., 2002; Gamper and Shapiro, 2003). In mammals, the CaM protein is coded by a multigene family consisting of three bona fide members, CALM1, CALM2 and CALM3. Within the non-coding regions of the CaM transcripts, no striking homology is observed, and codon usage is maximally divergent amongst the three CaM mRNAs that encode an identical protein. It has been hypothesised that the existence of a multigene family provides a tight and complex level of regulatory control at the level of gene expression (Palfi et al., 2002). CaM genes are differentially expressed in the CNS during development and differential regulation of the CaM genes appears necessary to maintain the temporal and spatial fidelity of the CaM protein levels in all subcellular domains. Besides the fundamental housekeeping functions associated with CaM, it is also involved in specialized neuronal functions, such as the synthesis and release of neurotransmitters, neurite extension, long-term potentiation and axonal transport (Palfi et al., 2002).

#### B) Effect of epilepsy-associated KCNQ2 mutations on the CaM-KCNQ2 interaction

To assess the effect that the C-terminus mutations of the present invention had on CaM binding, two of the identified mutations (R353G and L619R) were introduced into the DB-Q2C construct by mutagenesis and were re-analysed for an interaction with CaM using the yeast two-hybrid procedure.

The following primers were used to incorporate the c1057C→G (R353G) and c1856T→G (L619R) changes into the pDEST<sup>TM</sup>32- KCNQ2 C-terminal bait construct.

R353G F 5'-CGCCACCAACCTCTCGGGCACAGACCTGCACTC-3'

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R353G R 5'-GAGTGCAGGTCTGTGCCCCGAGAGGTTGGTGGCG-3'

L619R F 5'-CTTGTCCATGGAGAAGAAGCGGGACTTCCTGGTGAATATC-3'

L619R R 5'-GATATTCACCAGGAAGTCCCGCTTCTTCTCCATGGACAAG-3'

5        Overlapping PCR products were generated using the  
TOPO<sup>®</sup> cloning compatible KCNQ2F primer from the initial  
cloning and the mutagenesis reverse primers, and the  
KCNQ2R primer from the initial cloning with the  
mutagenesis forward primers. Products were gel extracted  
10 and purified before a second round of PCR using the  
initial KCNQ2 F&R primers. These products were also gel  
extracted before cloning into the pDEST<sup>™</sup>32 bait vector via  
the TOPO<sup>®</sup> system (as described above). Mutant baits were  
sequence verified.

15        The interaction between each DB-Q2C mutant and CaM  
was then tested by the yeast two-hybrid assay and compared  
to the interaction with DB-Q2 wt. Three different PCR-  
amplified CaM positive clones from the initial screen were  
re-introduced by gap-repair<sup>20</sup> into the prey vector (pPC86)  
20 in the yeast strain expressing either DB-Q2C wt, DB-Q2C  
mutants or the empty DBLeu vector, used as negative  
control.

CaM interaction with the DB-Q2C wt and mutants was  
then assessed by expression of the *HIS3* and *LacZ* reporter  
25 genes.

The Q2C R353G mutant did not interact with CaM, as  
seen by no growth on *HIS3* selective plate (Figure 4C) and  
no blue readout in the *LacZ* filter assay (seen as dark  
squares in Figure 4D-F). On the other hand, the DB-Q2C  
30 L619R mutant was shown to still interact with CaM, as seen  
by growth on *HIS3* selective plate (Figure 4C) and the blue  
readout in the *LacZ* filter assay. Interestingly, the DB-  
Q2C L619R mutant showed an even greater growth level on  
*HIS3* selective plate than the DB-Q2C wt and also appeared  
35 to stain faster and more intensely blue in the *LacZ* filter  
assay, suggesting a stronger interaction between CaM and  
this mutant.

In order to better quantify  $\beta$ -gal activity, a second assay was carried out using the high sensitivity substrate Chlorophenol Red- $\beta$ -D-Galactopyranoside (CPRG) in liquid culture. The affinity of the DB-Q2C/AD-CaM interaction was measured in terms of units of  $\beta$ -gal activity, with a zero value indicating no expression of the *LacZ* reporter gene, and hence no interaction.

In the CPRG assay, a value of 0.05 units  $\beta$ -gal activity (Figure 5) was significantly different from the empty bait vector replicate ( $P < 0.01$ , Student's *t* test), confirming the interaction of the DB-Q2C wt with CaM.

As observed in the *LacZ* filter assay, the CPRG assay showed a significant difference in the interaction between the Q2C R353G mutant and CaM as compared to the wt replicate ( $P < 0.01$ , Student's *t* test, Figure 4).

These results suggest that the R353G mutation alters the structural conformation of the KCNQ2 C-terminal domain such that it is no longer able to bind to CaM and that this single point mutation is sufficient to abolish the interaction. By abolishing CaM binding, the R353G mutation could lead to an impairment of M-current *in vivo* due to decreased opening of the channel.

In contrast, the CPRG assay for the L619R Q2C mutant showed a significantly higher level of  $\beta$ -gal activity units (0.26 units) than the wt replicate ( $P < 0.001$ , Student's *t* test, Figure 5). This finding indicates that the L619R mutation alters the conformation of the protein in a manner that increases CaM binding affinity for the KCNQ2 C-terminal domain by approximately 5-fold. The increased affinity for CaM may affect the ability of the complex to change conformation normally in response to calcium signalling. Alternatively, the marked increase in binding of CaM to the KCNQ2 L619R mutant channel may be detrimental to the M-channel function via disruption of the normal neuronal inhibitory/excitatory balance, therefore causing the seizures associated with epilepsy, particularly BFNS. CaM is known to be involved in both the

excitatory and inhibitory neurotransmission pathways (Ohya and Botstein, 1994) and it has been proposed that the temporal and spatial restrictions on CaM itself could enable the tight control of these opposing reactions (Toutenhoofd and Strehler, 2000). Hence, the KCNQ2 L619R mutation could lead to a disruption of the local CaM pool consequently disturbing the finely balanced excitatory and inhibitory neurotransmission systems.

These results implicate CaM in the pathogenesis of epilepsy and specifically in the BFNS syndrome. Whilst further work will be required to fully elucidate the involvement of the KCNQ2-CaM interaction in neuronal excitability and its correlation with idiopathic epilepsy, these data suggest that dysfunction of this interaction leads to aberrant neuronal excitability in some BFNS patients.

The calmodulin gene (and other ion channel interacting genes) may therefore be a target for mutation in epilepsy as well as other disorders associated with ion channel dysfunction. A mutation in an ion channel interacting gene when expressed alone, or when expressed in combination with one or more other ion channel mutations or ion channel interacting gene mutations (based on the digenic model), may give rise to the disorder. The nature of the ion channel interacting genes and proteins can be studied such that these partners can also be targets for drug discovery.

#### Industrial Applicability

The mutant ion channel receptor subunits of the invention are useful in the diagnosis and treatment of diseases such as epilepsy and disorders associated with ion channel dysfunction including, but not limited to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia,

anxiety, depression, phobic obsessive symptoms,  
neuropathic pain, inflammatory pain, chronic/acute pain,  
Bartter's syndrome, polycystic kidney disease, Dent's  
disease, hyperinsulinemic hypoglycemia of infancy, cystic  
5 fibrosis, congenital stationary night blindness and total  
colour-blindness.

TABLE 1

Examples of mutations and variations identified in ion channel subunit genes

Subunit Gene	Exon/Intron	DNA Mutation	Amino Acid Change	SEQ ID NOS
<b>Sodium Channel Subunits</b>				
Coding exonic variants – amino acid change				
SCN1A <sup>r</sup>	Exon 5	c664C→T	R222X	1, 73
SCN1A <sup>r</sup>	Exon 8	c1152G→A	W384X	2, 74
SCN1A <sup>r</sup>	Exon 9	c1183G→C	A395P	3, 75
SCN1A <sup>r</sup>	Exon 9	c1207T→C	F403L	4, 76
SCN1A <sup>r</sup>	Exon 9	c1237T→A	Y413N	5, 77
SCN1A <sup>r</sup>	Exon 9	c1265T→A	V422E	6, 78
SCN1A <sup>r</sup>	Exon 21	c4219C→T	R1407X	7, 79
SCN1A <sup>r</sup>	Exon 26	c5339T→C	M1780T	8, 80
SCN1A <sup>r</sup>	Exon 26	c5674C→T	R1892X	9, 81
SCN1B <sup>r</sup>	Exon 3	c254G→A	R85H	10, 82
SCN2A <sup>r</sup>	Exon 6A	c668G→A	R223Q	11, 83
SCN2A <sup>r</sup>	Exon 16	c2674G→A	V892I	12, 84
SCN2A <sup>r</sup>	Exon 17	c3007C→A	L1003I	13, 85
SCN2A <sup>r</sup>	Exon 19	c3598A→G	T1200A	14, 86
SCN2A <sup>r</sup>	Exon 20	c3956G→A	R1319Q	15, 87
Coding exonic variants – no amino acid change				
SCN2A <sup>c</sup>	Exon 12	c1785T→C	-	16
SCN2A <sup>c</sup>	Exon 27	c4919T→A	-	17
Non-coding variants				
SCN1A <sup>r</sup>	Intron 9	IVS9-1G→A	-	18
SCN1A <sup>c</sup>	Intron 23	IVS23+33G→A	-	19
SCN2A <sup>r</sup>	Intron 7	IVS7+61T→A	-	20
SCN2A <sup>r</sup>	Intron 19	IVS19-55A→G	-	21
SCN2A <sup>r</sup>	Intron 22	IVS22-31A→G	-	22
SCN2A <sup>c</sup>	Intron 2	IVS2-28G→A	-	23
SCN2A <sup>c</sup>	Intron 8	IVS8-3T→C	-	24
SCN2A <sup>c</sup>	Intron 11	IVS11+49A→G	-	25
SCN2A <sup>c</sup>	Intron 11	IVS11-16C→T	-	26
SCN2A <sup>c</sup>	Intron 17	IVS17-71C→T	-	27
SCN2A <sup>c</sup>	Intron 17	IVS17-74delG	-	28
SCN2A <sup>c</sup>	Intron 17	IVS17-74insG	-	29
<b>Nicotinic Acetylcholine Receptor Subunits</b>				
Coding exonic variants – amino acid change				
CHRNA5 <sup>r</sup>	Exon 4	c400G→A	V134I	30, 88
CHRNA2 <sup>c</sup>	Exon 4	c373G→A	A125T	31, 89
CHRNA3 <sup>c</sup>	Exon 2	c110G→A	R37H	32, 90
Coding variants – no amino acid change				
CHRNA2 <sup>c</sup>	Exon 4	c351C→T	-	33
CHRNA2 <sup>c</sup>	Exon 5	c771C→T	-	34
CHRNA3 <sup>c</sup>	Exon 2	c159A→G	-	35
CHRNA3 <sup>c</sup>	Exon 4	c291G→A	-	36
CHRNA3 <sup>c</sup>	Exon 4	c345G→A	-	37

TABLE 1 (Continued)

Examples of mutations and variations identified in ion channel subunit genes				
Subunit Gene	Exon/Intron	DNA Mutation	Amino Acid Change	SEQ ID NOS
<b>Non-coding variants</b>				
CHRNA2 <sup>c</sup>	Intron 3	IVS3-16C→T	-	38
CHRNA3 <sup>c</sup>	Intron 3	IVS3-5T→C	-	39
CHRNA3 <sup>c</sup>	Intron 4	IVS4+8G→C	-	40
<b>Potassium Channel Subunits</b>				
<b>Coding exonic variants – amino acid change</b>				
KCNQ2 <sup>r</sup>	Exon 1	c204-c205insC	K69fsX119	41, 91
KCNQ2 <sup>r</sup>	Exon 1	c1A→G	M1V	42
KCNQ2 <sup>r</sup>	Exon 1	c2T→C	M1T	43
KCNQ2 <sup>r</sup>	Exon 8	c1057C→G	R353G	44, 92
KCNQ2 <sup>r</sup>	Exon 11	c1288C→T	R430X	45, 93
KCNQ2 <sup>r</sup>	Exon 14	c1710A→T	R570S	46, 94
KCNQ2 <sup>r</sup>	Exon 15	c1856T→G	L619R	47, 95
<b>Non-coding variants</b>				
KCNQ2 <sup>r</sup>	Intron 9	IVS9+(46-48)delCCT	-	48
KCNQ3 <sup>r</sup>	Intron 11	IVS11+43G→A	-	49
KCNQ3 <sup>c</sup>	Intron 12	IVS12+29G→A	-	50
<b>GABA Receptor Subunits</b>				
<b>Coding exonic variants – no amino acid change</b>				
GABRB1 <sup>r</sup>	Exon 5	c508C→T	-	51
GABRB1 <sup>r</sup>	Exon 9	c1329G→A	-	52
GABRB1 <sup>c</sup>	Exon 8	c975C→T	-	53
GABRG3 <sup>c</sup>	Exon 8	c995T→C	-	54
<b>Non-coding variants</b>				
GABRA1 <sup>c</sup>	5' UTR	c-142A→G	-	55
GABRA1 <sup>c</sup>	5' UTR	c-31C→T	-	56
GABRA2 <sup>c</sup>	3' UTR	c1615G→A	-	57
GABRA5 <sup>c</sup>	5' UTR	c-271G→C	-	58
GABRA5 <sup>c</sup>	5' UTR	c-228A→G	-	59
GABRA5 <sup>c</sup>	5' UTR	c-149G→C	-	60
GABRB2 <sup>b</sup>	5' UTR	c-159C→T	-	61
GABRB2 <sup>c</sup>	3' UTR	c1749C→T	-	62
GABRP1 <sup>c</sup>	5' UTR	c-101C→T	-	63
GABRB1 <sup>c</sup>	Intron 1	IVS1+24T→G	-	64
GABRB1 <sup>c</sup>	Intron 5	IVS6+72T→G	-	65
GABRB1 <sup>c</sup>	Intron 7	IVS7-34A→G	-	66
GABRB3 <sup>r</sup>	Intron 1	IVS1-14C→T	-	67
GABRB3 <sup>r</sup>	Intron 7	IVS7+58delA	-	68
GABRD <sup>r</sup>	Intron 6	IVS6+132insC	-	69
GABRD <sup>r</sup>	Intron 6	IVS6+130insC	-	70
GABRD <sup>r</sup>	Intron 6	IVS6+73del	-	71
CGCGCCACCGCCCCCTTCCGCG				
GABRG3 <sup>c</sup>	Intron 8	IVS8-102C→T	-	72

Note: <sup>r</sup> Mutations or variations only occurring in individuals with epilepsy; <sup>b</sup> Variant seen only in normal control samples; <sup>c</sup> Mutations or variants seen in individuals with epilepsy as well as normal control samples. The KCNQ2 numbering is based on the large isoform (inclusion of exon 10a). The numbering of exons and introns for SCN2A is based on the publication of Kassai et al., 2001.



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Claims

1. A method of identifying a subject predisposed to a disorder associated with ion channel dysfunction, comprising ascertaining whether at least one of the genes encoding ion channel subunits in said subject has undergone a mutation event selected from the group consisting of the mutation events set forth in the following Table:

Subunit Gene	Exon/Intron	DNA Mutation
SCN1A	Exon 5	c664C→T
SCN1A	Exon 8	c1152G→A
SCN1A	Exon 9	c1183G→C
SCN1A	Exon 9	c1207T→C
SCN1A	Exon 9	c1237T→A
SCN1A	Exon 9	c1265T→A
SCN1A	Exon 21	c4219C→T
SCN1A	Exon 26	c5339T→C
SCN1A	Exon 26	c5674C→T
SCN1B	Exon 3	c254G→A
SCN2A	Exon 6A	c668G→A
SCN2A	Exon 16	c2674G→A
SCN2A	Exon 17	c3007C→A
SCN2A	Exon 19	c3598A→G
SCN2A	Exon 20	c3956G→A
SCN2A	Exon 12	c1785T→C
SCN2A	Exon 27	c4919T→A
SCN1A	Intron 9	IVS9-1G→A
SCN1A	Intron 23	IVS23+33G→A
SCN2A	Intron 7	IVS7+61T→A
SCN2A	Intron 19	IVS19-55A→G
SCN2A	Intron 22	IVS22-31A→G
SCN2A	Intron 2	IVS2-28G→A
SCN2A	Intron 8	IVS8-3T→C
SCN2A	Intron 11	IVS11+49A→G
SCN2A	Intron 11	IVS11-16C→T
SCN2A	Intron 17	IVS17-71C→T
SCN2A	Intron 17	IVS17-74delG
SCN2A	Intron 17	IVS17-74insG
CHRNA5	Exon 4	c400G→A
CHRNA2	Exon 4	c373G→A
CHRNA3	Exon 2	c110G→A
CHRNA2	Exon 4	c351C→T

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CHRNA2	Exon 5	c771C→T
CHRNA3	Exon 2	c159A→G
CHRNA3	Exon 4	c291G→A
CHRNA3	Exon 4	c345G→A
CHRNA2	Intron 3	IVS3-16C→T
CHRNA3	Intron 3	IVS3-5T→C
CHRNA3	Intron 4	IVS4+8G→C
KCNQ2	Exon 1	c204-c205insC
KCNQ2	Exon 1	c1A→G
KCNQ2	Exon 1	c2T→C
KCNQ2	Exon 8	c1057C→G
KCNQ2	Exon 11	c1288C→T
KCNQ2	Exon 14	c1710A→T
KCNQ2	Exon 15	c1856T→G
KCNQ2	Intron 9	IVS9+ (46-48) delCCT
KCNQ3	Intron 11	IVS11+43G→A
KCNQ3	Intron 12	IVS12+29G→A
GABRB1	Exon 5	c508C→T
GABRB1	Exon 9	c1329G→A
GABRB1	Exon 8	c975C→T
GABRG3	Exon 8	c995T→C
GABRA1	5' UTR	c-142A→G
GABRA1	5' UTR	c-31C→T
GABRA2	3' UTR	c1615G→A
GABRA5	5' UTR	c-271G→C
GABRA5	5' UTR	c-228A→G
GABRA5	5' UTR	c-149G→C
GABRB2	5' UTR	c-159C→T
GABRB2	3' UTR	c1749C→T
GABRPi	5' UTR	c-101C→T
GABRB1	Intron 1	IVS1+24T→G
GABRB1	Intron 6	IVS6+72T→G
GABRB1	Intron 7	IVS7-34A→G
GABRB3	Intron 1	IVS1-14C→T
GABRB3	Intron 7	IVS7+58delAA
GABRD	Intron 6	IVS6+132insC
GABRD	Intron 6	IVS6+130insC
GABRD	Intron 6	IVS6+73delCGCGCCACCGCCCCTTCCGCG
GABRG3	Intron 8	IVS8-102C→T

2. A method as claimed in claim 1 wherein a cDNA derived from said subject comprises the sequence set forth in one of SEQ ID NOS: 1-72.

3. A method as claimed in claim 1 wherein a cDNA derived from said subject has the sequence set forth in one of SEQ ID NOS: 1-72.

4. A method as claimed in any one of claims 1 to 3, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce an epilepsy phenotype in said subject.

5. A method as claimed in any one of claims 1 to 3, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce one or more disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness in said subject.

6. A method as claimed in any one of claims 1 to 3, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce an epilepsy phenotype when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

7. A method as claimed in any one of claims 1 to 3, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce one or more disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia,



myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness, when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

8. An isolated nucleic acid molecule encoding a mutant or variant ion channel subunit wherein a mutation event selected from the group consisting of the mutation events set forth in the following Table:

Subunit Gene	Exon/Intron	DNA Mutation
SCN1A	Exon 5	c664C→T
SCN1A	Exon 8	c1152G→A
SCN1A	Exon 9	c1183G→C
SCN1A	Exon 9	c1207T→C
SCN1A	Exon 9	c1237T→A
SCN1A	Exon 9	c1265T→A
SCN1A	Exon 21	c4219C→T
SCN1A	Exon 26	c5339T→C
SCN1A	Exon 26	c5674C→T
SCN1B	Exon 3	c254G→A
SCN2A	Exon 6A	c668G→A
SCN2A	Exon 16	c2674G→A
SCN2A	Exon 17	c3007C→A
SCN2A	Exon 19	c3598A→G
SCN2A	Exon 20	c3956G→A
SCN2A	Exon 12	c1785T→C
SCN2A	Exon 27	c4919T→A
SCN1A	Intron 9	IVS9-1G→A
SCN1A	Intron 23	IVS23+33G→A
SCN2A	Intron 7	IVS7+61T→A
SCN2A	Intron 19	IVS19-55A→G
SCN2A	Intron 22	IVS22-31A→G
SCN2A	Intron 2	IVS2-28G→A
SCN2A	Intron 8	IVS8-3T→C
SCN2A	Intron 11	IVS11+49A→G
SCN2A	Intron 11	IVS11-16C→T

SCN2A	Intron 17	IVS17-71C→T
SCN2A	Intron 17	IVS17-74delG
SCN2A	Intron 17	IVS17-74insG
CHRNA5	Exon 4	c400G→A
CHRNA2	Exon 4	c373G→A
CHRNA3	Exon 2	c110G→A
CHRNA2	Exon 4	c351C→T
CHRNA2	Exon 5	c771C→T
CHRNA3	Exon 2	c159A→G
CHRNA3	Exon 4	c291G→A
CHRNA3	Exon 4	c345G→A
CHRNA2	Intron 3	IVS3-16C→T
CHRNA3	Intron 3	IVS3-5T→C
CHRNA3	Intron 4	IVS4+8G→C
KCNQ2	Exon 1	c204-c205insC
KCNQ2	Exon 1	c1A→G
KCNQ2	Exon 1	c2T→C
KCNQ2	Exon 8	c1057C→G
KCNQ2	Exon 11	c1288C→T
KCNQ2	Exon 14	c1710A→T
KCNQ2	Exon 15	c1856T→G
KCNQ2	Intron 9	IVS9+(46-48)delCCT
KCNQ3	Intron 11	IVS11+43G→A
KCNQ3	Intron 12	IVS12+29G→A
GABRB1	Exon 5	c508C→T
GABRB1	Exon 9	c1329G→A
GABRB1	Exon 8	c975C→T
GABRG3	Exon 8	c995T→C
GABRA1	5' UTR	c-142A→G
GABRA1	5' UTR	c-31C→T
GABRA2	3' UTR	c1615G→A
GABRA5	5' UTR	c-271G→C
GABRA5	5' UTR	c-228A→G
GABRA5	5' UTR	c-149G→C
GABRB2	5' UTR	c-159C→T
GABRB2	3' UTR	c1749C→T
GABRPi	5' UTR	c-101C→T
GABRB1	Intron 1	IVS1+24T→G
GABRB1	Intron 6	IVS6+72T→G
GABRB1	Intron 7	IVS7-34A→G
GABRB3	Intron 1	IVS1-14C→T
GABRB3	Intron 7	IVS7+58delAA
GABRD	Intron 6	IVS6+132insC
GABRD	Intron 6	IVS6+130insC
GABRD	Intron 6	IVS6+73delCGCGCCACCGCCCTTCCGCG
GABRG3	Intron 8	IVS8-102C→T

---

has occurred.

9. An isolated nucleic acid molecule encoding a mutant  
or variant ion channel subunit as claimed in claim 8  
5 wherein a cDNA derived therefrom comprises the sequence  
set forth in one of SEQ ID NOS: 1-72.

10. An isolated nucleic acid molecule encoding a mutant  
or variant ion channel subunit as claimed in claim 8  
10 wherein a cDNA derived therefrom has the sequence set  
forth in one of SEQ ID NOS: 1-72.

11. An isolated nucleic acid molecule encoding a mutant  
or variant ion channel subunit as claimed in any one of  
15 claims 8 to 10, wherein said mutation event disrupts the  
functioning of an assembled ion channel so as to produce  
an epilepsy phenotype.

12. An isolated nucleic acid molecule encoding a mutant  
20 or variant ion channel subunit as claimed in any one of  
claims 8 to 10, wherein said mutation event disrupts the  
functioning of an assembled ion channel so as to produce  
one or more disorders associated with ion channel  
dysfunction, including but not restricted to, hyper- or  
25 hypo-kalemic periodic paralysis, myotonias, malignant  
hyperthermia, myasthenia, cardiac arrhythmias, episodic  
ataxia, migraine, Alzheimer's disease, Parkinson's  
disease, schizophrenia, hyperekplexia, anxiety,  
depression, phobic obsessive symptoms, neuropathic pain,  
30 inflammatory pain, chronic/acute pain, Bartter's syndrome,  
polycystic kidney disease, Dent's disease,  
hyperinsulinemic hypoglycemia of infancy, cystic fibrosis,  
congenital stationary night blindness and total colour-  
blindness.

35

13. An isolated nucleic acid molecule encoding a mutant  
or variant ion channel subunit as claimed in any one of

claims 8 to 10, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce an epilepsy phenotype when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

14. An isolated nucleic acid molecule encoding a mutant or variant ion channel subunit as claimed in any one of claims 8 to 10, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce one or more disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness, when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

25

15. An isolated nucleic acid molecule comprising any one of the nucleotide sequences set forth in SEQ ID NOS: 1-72.

16. An isolated nucleic acid molecule consisting of any one of the nucleotide sequences set forth in SEQ ID NOS: 1-72.

17. An isolated nucleic acid molecule encoding a mutant KCNQ2 subunit, wherein the mutation event has occurred in the C-terminal domain of the KCNQ2 subunit and leads to a disturbance in the calmodulin binding affinity of the subunit, so as to produce an epilepsy phenotype.

18. An isolated nucleic acid molecule as claimed in claim 17 wherein the mutation event has occurred in exon 8, exon 11, exon 14 or exon 15.

5

19. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit wherein a mutation event selected from the group consisting of the mutation events set forth in the following Table:

Subunit Gene	Amino Acid Change
SCN1A	R222X
SCN1A	W384X
SCN1A	A395P
SCN1A	F403L
SCN1A	Y413N
SCN1A	V422E
SCN1A	R1407X
SCN1A	M1780T
SCN1A	R1892X
SCN1B	R85H
SCN2A	R223Q
SCN2A	V892I
SCN2A	L1003I
SCN2A	T1200A
SCN2A	R1319Q
CHRNA5	V134I
CHRNA2	A125T
CHRNA3	R37H
KCNQ2	K69fsX119
KCNQ2	M1V
KCNQ2	M1T
KCNQ2	R353G
KCNQ2	R430X
KCNQ2	R570S
KCNQ2	L619R

10 has occurred.

20. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit as claimed in claim 19 wherein the polypeptide comprises the amino acid sequence set forth in one of SEQ ID NOS: 73-95.

15

21. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit as claimed in claim

19 wherein the polypeptide has the amino acid sequence set forth in one of SEQ ID NOS: 73-95.

22. An isolated polypeptide, said polypeptide being a  
5 mutant or variant ion channel subunit as claimed in any one of claims 19 to 21, wherein said mutation event disrupts the functioning of an assembled ion channel so as to produce an epilepsy phenotype.

10 23. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit as claimed in any one of claims 19 to 21, wherein said mutation event disrupts the functioning of an assembled ion channel so as  
15 to produce one or more disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia,  
20 anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total  
25 colour-blindness.

24. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit as claimed in any one of claims 19 to 21, wherein said mutation event  
30 disrupts the functioning of an assembled ion channel so as to produce an epilepsy phenotype when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

35 25. An isolated polypeptide, said polypeptide being a mutant or variant ion channel subunit as claimed in any one of claims 19 to 21, wherein said mutation event

disrupts the functioning of an assembled ion channel so as to produce one or more disorders associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness and total colour-blindness, when expressed in combination with one or more additional mutations or variations in said ion channel subunit genes.

26. An isolated polypeptide comprising any one of the amino acid sequences set forth in SEQ ID NOS: 73-95.

27. An isolated polypeptide consisting of any one of the amino acid sequences set forth in SEQ ID NOS: 73-95.

28. An isolated polypeptide, said polypeptide being a mutant KCNQ2 subunit, wherein the mutation event has occurred in the C-terminal domain of the KCNQ2 subunit and leads to a disturbance in the calmodulin binding affinity of the subunit, so as to produce an epilepsy phenotype.

29. An isolated polypeptide complex, said polypeptide complex being an assembled mammalian ion channel including an ion channel subunit comprising a polypeptide as defined in any one of claims 19 to 28.

30. An expression vector comprising a nucleic acid molecule as claimed in any one of claims 8 to 18.

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31. A cell comprising at least one expression vector as claimed in claim 30.

5 32. A cell as claimed in claim 31 comprising two or more expression vectors.

10 33. A cell comprising at least one ion channel type, wherein the or each ion channel type incorporates at least one mutant polypeptide as claimed in any one claims 19 to 28.

34. A cell as claimed in claim 33 comprising ion channels that incorporate two or more mutant polypeptides.

15 35. A cell as claimed in claim 33 comprising two or more ion channel types each incorporating one or more mutant polypeptides.

20 36. A method of preparing a polypeptide, comprising the steps of:

(1) culturing cells as claimed in any one of claims 31 to 35 under conditions effective for polypeptide production; and

(2) harvesting the polypeptide.

25

37. A polypeptide prepared by the method of claim 36.

30 38. An antibody which is immunologically reactive with an isolated polypeptide as claimed in any one of claims 19 to 28 or claim 37, or an isolated polypeptide complex as claimed in claim 29.

35 39. An antibody as claimed in claim 38 which is selected from the group consisting of a monoclonal antibody, a humanised antibody, a chimeric antibody or an antibody fragment including a Fab fragment, (Fab')<sub>2</sub> fragment, Fv



fragment, single chain antibodies and single domain antibodies.

40. A method of treating epilepsy comprising  
5 administering an antibody as claimed in either one of  
claims 38 or 39 to a subject in need of such treatment.

41. The use of an antibody, as claimed in either one of  
claims 38 or 39, in the manufacture of a medicament for  
10 the treatment of epilepsy.

42. A method of treating a disorder associated with ion  
channel dysfunction, including but not restricted to,  
hyper- or hypo-kalemic periodic paralysis, myotonias,  
15 malignant hyperthermia, myasthenia, cardiac arrhythmias,  
episodic ataxia, migraine, Alzheimer's disease,  
Parkinson's disease, schizophrenia, hyperekplexia,  
anxiety, depression, phobic obsessive symptoms,  
neuropathic pain, inflammatory pain, chronic/acute pain,  
20 Bartter's syndrome, polycystic kidney disease, Dent's  
disease, hyperinsulinemic hypoglycemia of infancy, cystic  
fibrosis, congenital stationary night blindness or total  
colour-blindness, comprising administering an antibody as  
claimed in either one of claims 38 or 39 to a subject in  
25 need of such treatment.

43. The use of an antibody, as claimed in either one of  
claims 38 or 39, in the manufacture of a medicament for  
the treatment of a disorder associated with ion channel  
30 dysfunction, including but not restricted to, hyper- or  
hypo-kalemic periodic paralysis, myotonias, malignant  
hyperthermia, myasthenia, cardiac arrhythmias, episodic  
ataxia, migraine, Alzheimer's disease, Parkinson's  
disease, schizophrenia, hyperekplexia, anxiety,  
35 depression, phobic obsessive symptoms, neuropathic pain,  
inflammatory pain, chronic/acute pain, Bartter's syndrome,  
polycystic kidney disease, Dent's disease,

hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

5 44. A method of treating epilepsy comprising administering a selective agonist, antagonist or modulator of an ion channel when it has undergone a mutation event or combination of events as defined in any one of claims 19 to 28 to a subject in need of such treatment.

10 45. The use of a selective agonist, antagonist or modulator of an ion channel when it has undergone a mutation event as defined in any one of claims 19 to 28 in the manufacture of a medicament for the treatment of  
15 epilepsy.

46. A method of treating a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias,  
20 malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain,  
25 Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, comprising administering a selective agonist, antagonist or modulator of an ion channel when it  
30 has undergone a mutation event or combination of events as defined in any one of claims 19 to 28 to a subject in need of such treatment.

47. The use of a selective agonist, antagonist or  
35 modulator of an ion channel when it has undergone a mutation event as claimed in any one of claims 19 to 28 in the manufacture of a medicament for the treatment of a

disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

48. A method of treating epilepsy comprising administering an isolated DNA molecule which is the complement (antisense) of a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, to a subject in need of such treatment.

49. The use of a DNA molecule which is the complement of a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, in the manufacture of a medicament for the treatment of epilepsy.

50. A method of treating a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic

fibrosis, congenital stationary night blindness or total colour-blindness, comprising administering an isolated DNA molecule which is the complement (antisense) of a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, to a subject in need of such treatment.

51. The use of a DNA molecule which is the complement of a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, in the manufacture of a medicament for the treatment of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

52. A method of treating epilepsy comprising administering an antibody, as claimed in either one of claims 38 or 39, administration of an agonist, antagonist or modulator of an ion channel when it has undergone a mutation event or combination of events as defined in any one of claims 19 to 28, or administration of a DNA molecule which is the complement of a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one

of claims 8 to 18, in combination with administration of the wild-type ion channel subunit, to a subject in need of such treatment.

5 53. The use of an antibody, as claimed in claims 38 or 39, use of an agonist, antagonist or modulator of an ion channel when it has undergone a mutation event or combination of events as defined in any one of claims 19 to 28, or use of a DNA molecule which is the complement of  
10 a nucleic acid molecule as claimed in any one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, in combination with the use of the wild-type ion channel subunit, in the  
15 manufacture of a medicament for the treatment of epilepsy.

54. A method of treating a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias,  
20 malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain,  
25 Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, comprising administering an antibody, as claimed in either one of claims 38 or 39,  
30 administration of an agonist, antagonist or modulator of an ion channel when it has undergone a mutation event or combination of events as defined in any one of claims 19 to 28, or administration of a DNA molecule which is the complement of a nucleic acid molecule as claimed in any  
35 one of claims 8 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, in

combination with administration of the wild-type ion channel subunit, to a subject in need of such treatment.

55. The use of an antibody, as claimed in claims 387 or  
5 39, use of an agonist, antagonist or modulator of an ion channel when it has undergone a mutation event or combination of events as defined in any one of claims 19 to 28, or use of a DNA molecule which is the complement of a nucleic acid molecule as claimed in any one of claims 8  
10 to 18 and which encodes an RNA molecule that hybridizes with the mRNA encoded by a nucleic acid molecule as claimed in any one of claims 8 to 18, in combination with the use of the wild-type ion channel subunit, in the manufacture of a medicament for the treatment of a  
15 disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease,  
20 schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night  
25 blindness or total colour-blindness.

56. Use of a nucleic acid molecule as claimed in any one of claims 8 to 18 for the screening of candidate pharmaceutical agents.

57. Use of a nucleic acid molecule as claimed in any one of claims 8 to 18 for the screening of candidate pharmaceutical agents useful for the treatment of epilepsy.

58. Use of a nucleic acid molecule as claimed in any one of claims 8 to 18 for the screening of candidate

pharmaceutical agents useful for the treatment of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

59. Use of a polypeptide as claimed in any one of claims 19 to 28 or claim 37, or a polypeptide complex as claimed in claim 29 for the screening of candidate pharmaceutical agents.

60. Use of a polypeptide as claimed in any one of claims 19 to 28 or claim 37, or a polypeptide complex as claimed in claim 29 for the screening of candidate pharmaceutical agents useful for the treatment of epilepsy.

61. Use of a polypeptide as claimed in any one of claims 19 to 28 or claim 37, or a polypeptide complex as claimed in claim 29 for the screening of candidate pharmaceutical agents useful for the treatment of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic

fibrosis, congenital stationary night blindness or total colour-blindness.

62. Use of a cell as claimed in any one of claims 31 to 35 for the screening of candidate pharmaceutical agents.

63. Use of a cell as claimed in any one of claims 31 to 35 for the screening of candidate pharmaceutical agents useful for the treatment of epilepsy.

10

64. Use of a cell as claimed in any one of claims 31 to 35 for the screening of candidate pharmaceutical agents useful for the treatment of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

25

65. A compound when identified through a use as claimed in any one of claims 56 to 64.

30

66. A pharmaceutical composition comprising a compound as claimed in claim 65 and a pharmaceutically acceptable carrier.

35

67. A genetically modified non-human animal comprising an isolated nucleic acid molecule as claimed in any one of claims 8 to 18.



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68. A genetically modified, non-human animal which comprises two or more isolated nucleic acid molecules as claimed in any one of claims 8 to 18.

5 69. A genetically modified non-human animal as claimed in either one of claims 67 or 68 in which the animal is selected from the group consisting of rats, mice, hamsters, guinea pigs, rabbits, dogs, cats, goats, sheep, pigs and non-human primates such as monkeys and  
10 chimpanzees.

70. A method of producing a non-human transgenic animal comprising a combination of two or more ion channel mutations, comprising the steps of:

- 15 (1) creating a non-human transgenic animal comprising a first nucleic acid molecule as claimed in any one of claims 8 to 18;
- (2) creating one or more additional non-human, transgenic animals comprising a second nucleic  
20 acid molecule as claimed in any one of claims 8 to 18; and
- (3) conducting mating combinations so as to produce progeny containing combinations of two or more ion channel mutations which  
25 effectively mimic combinations of ion channel mutations responsible for human disease.

71. A non-human, transgenic animal produced by the process of claim 70.

30

72. The use of a genetically modified non-human animal as claimed in any one of claims 67 to 69 or a non-human transgenic animal as claimed in claim 71 in the screening of candidate pharmaceutical compounds.

35

73. The use of a genetically modified non-human animal as claimed in any one of claims 67 to 69 or a non-human

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transgenic animal as claimed in claim 71 in the screening of candidate pharmaceutical compounds useful in the treatment of epilepsy.

5 74. The use of a genetically modified non-human animal as claimed in any one of claims 67 to 69 or a non-human transgenic animal as claimed in claim 71 in the screening of candidate pharmaceutical compounds useful in the treatment of a disorder associated with ion channel  
10 dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety,  
15 depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-  
20 blindness.

75. The use of an isolated nucleic acid molecule as claimed in any one of claims 8 to 18 for the diagnosis or prognosis of epilepsy.

25 76. The use of an isolated nucleic acid molecule as claimed in any one of claims 8 to 18 for the diagnosis or prognosis of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or  
30 hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain,  
35 inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis,

congenital stationary night blindness or total colour-blindness.

77. The use of a polypeptide as defined in any one of  
5 claims 19 to 28 or claim 37, or polypeptide complex as  
claimed in claim 29 in the diagnosis or prognosis of  
epilepsy.

78. The use of a polypeptide as defined in any one of  
10 claims 19 to 28 or claim 37, or polypeptide complex as  
claimed in claim 29 in the diagnosis or prognosis of a  
disorder associated with ion channel dysfunction,  
including but not restricted to, hyper- or hypo-kalemic  
periodic paralysis, myotonias, malignant hyperthermia,  
15 myasthenia, cardiac arrhythmias, episodic ataxia,  
migraine, Alzheimer's disease, Parkinson's disease,  
schizophrenia, hyperekplexia, anxiety, depression, phobic  
obsessive symptoms, neuropathic pain, inflammatory pain,  
chronic/acute pain, Bartter's syndrome, polycystic kidney  
20 disease, Dent's disease, hyperinsulinemic hypoglycemia of  
infancy, cystic fibrosis, congenital stationary night  
blindness or total colour-blindness.

79. The use of an antibody as claimed in either one of  
25 claims 38 or 39 in the diagnosis or prognosis of epilepsy.

80. The use of an antibody as claimed in either one of  
claims 38 or 39 in the diagnosis or prognosis of a  
disorder associated with ion channel dysfunction,  
30 including but not restricted to, hyper- or hypo-kalemic  
periodic paralysis, myotonias, malignant hyperthermia,  
myasthenia, cardiac arrhythmias, episodic ataxia,  
migraine, Alzheimer's disease, Parkinson's disease,  
schizophrenia, hyperekplexia, anxiety, depression, phobic  
35 obsessive symptoms, neuropathic pain, inflammatory pain,  
chronic/acute pain, Bartter's syndrome, polycystic kidney  
disease, Dent's disease, hyperinsulinemic hypoglycemia of

infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness.

81. A method for the diagnosis or prognosis of epilepsy comprising the steps of:

- (1) obtaining DNA from a subject; and
- (2) comparing the DNA of one or more subunits of ion channels from said subject to the DNA of the corresponding native subunits;

wherein identification of one or more DNA molecules as claimed in any one of claims 8 to 18 is an indication of epilepsy, or a predisposition thereto.

82. A method for the diagnosis or prognosis of a disorder associated with ion channel dysfunction, including but not restricted to, hyper- or hypo-kalemic periodic paralysis, myotonias, malignant hyperthermia, myasthenia, cardiac arrhythmias, episodic ataxia, migraine, Alzheimer's disease, Parkinson's disease, schizophrenia, hyperekplexia, anxiety, depression, phobic obsessive symptoms, neuropathic pain, inflammatory pain, chronic/acute pain, Bartter's syndrome, polycystic kidney disease, Dent's disease, hyperinsulinemic hypoglycemia of infancy, cystic fibrosis, congenital stationary night blindness or total colour-blindness, comprising the steps of:

- (1) obtaining DNA from a subject; and
- (2) comparing the DNA of one or more subunits of ion channels from said subject to the DNA of the corresponding native subunits;

wherein identification of one or more DNA molecules as claimed in any one of claims 8 to 18 is an indication of the disorder, or a predisposition thereto.

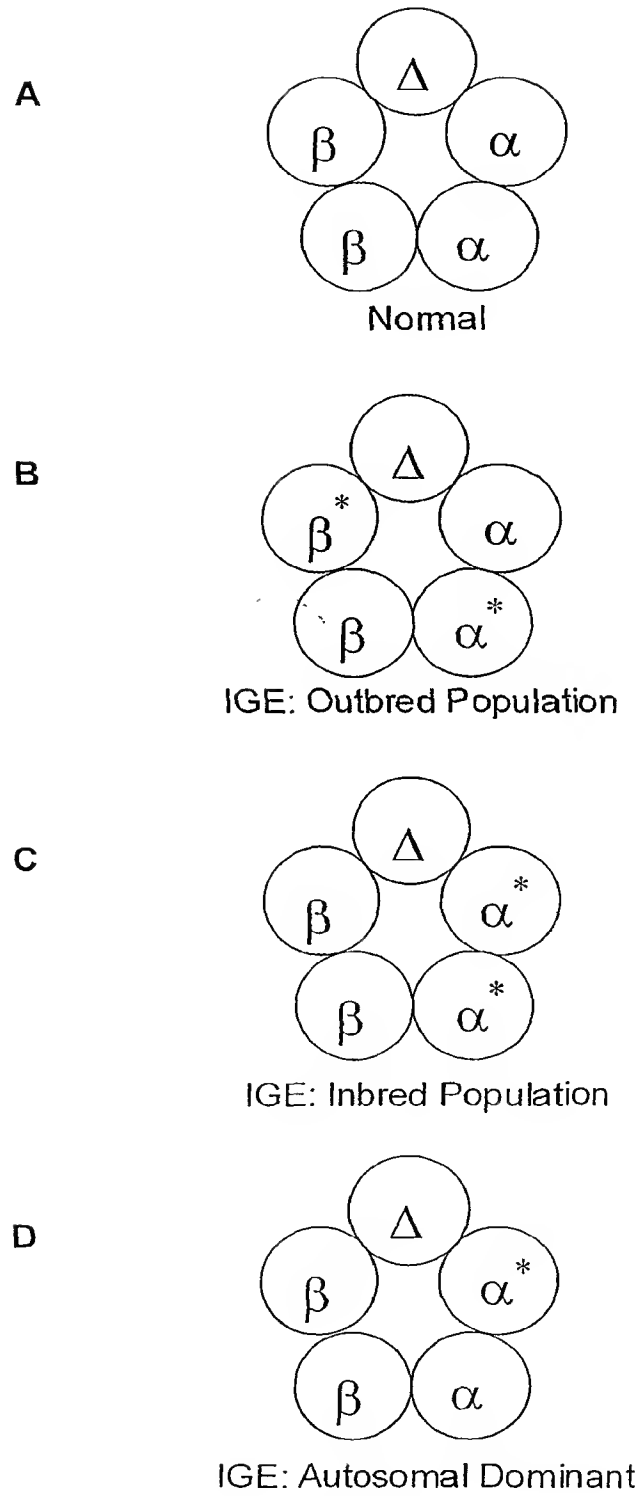
83. A method as claimed in either one of claims 81 or 82 wherein each DNA fragment is sequenced and the sequences compared.

84. A method as claimed in either one of claims 81 or 82 wherein the DNA fragments are subjected to restriction enzyme analysis.

5

85. A method as claimed in either one of claims 81 or 82 wherein the DNA fragments are subjected to SSCP analysis.

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**FIG. 1**

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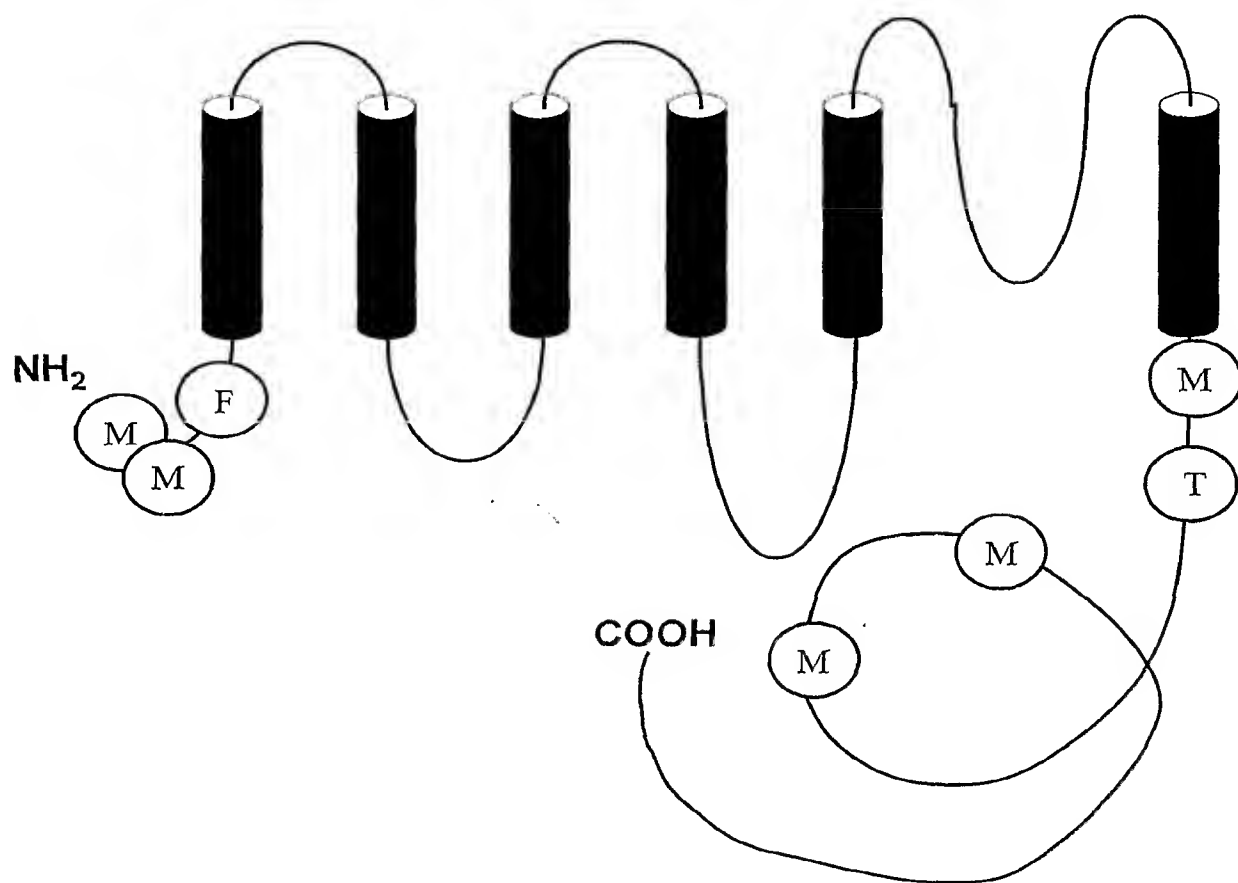
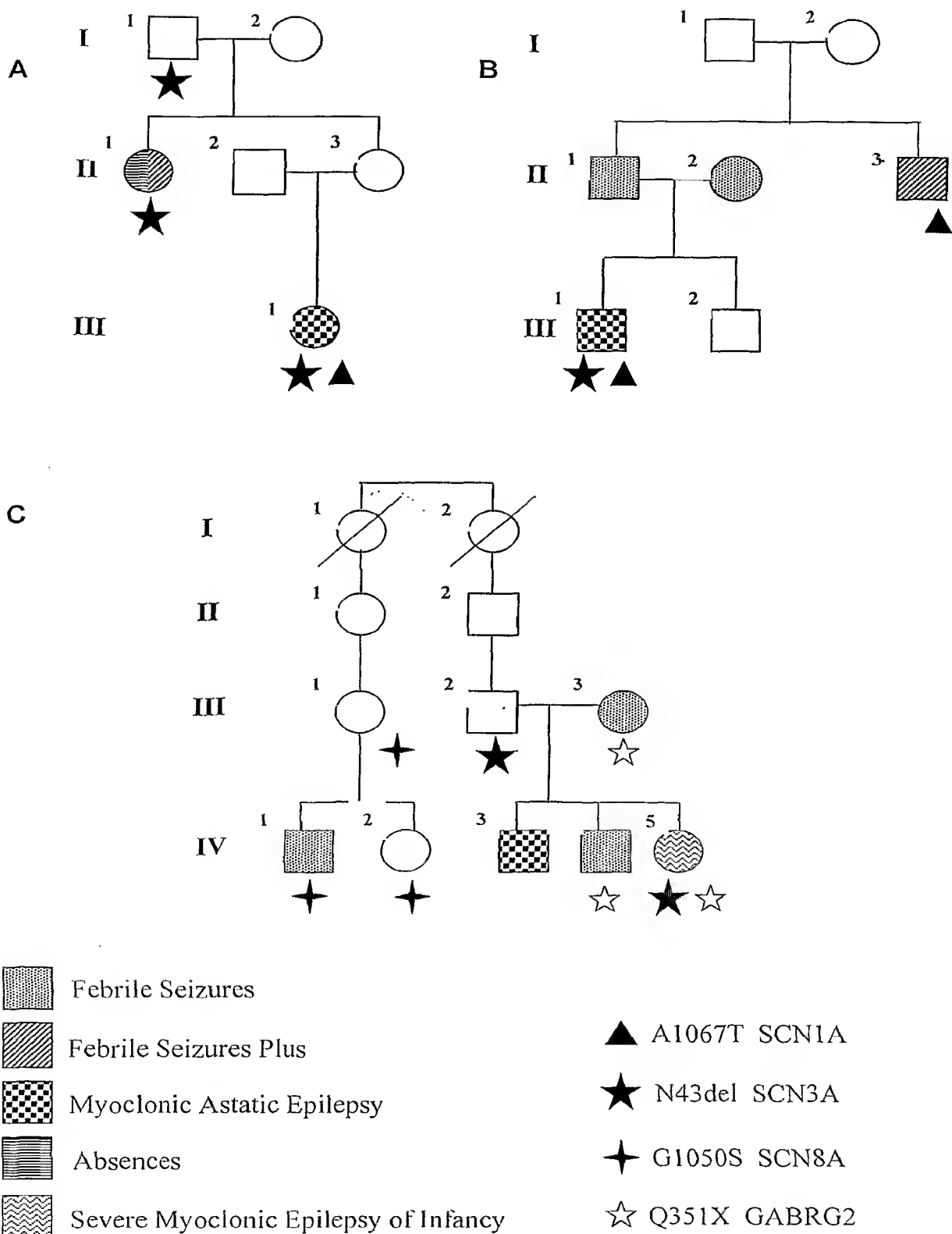


FIG. 2

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**FIG. 3**



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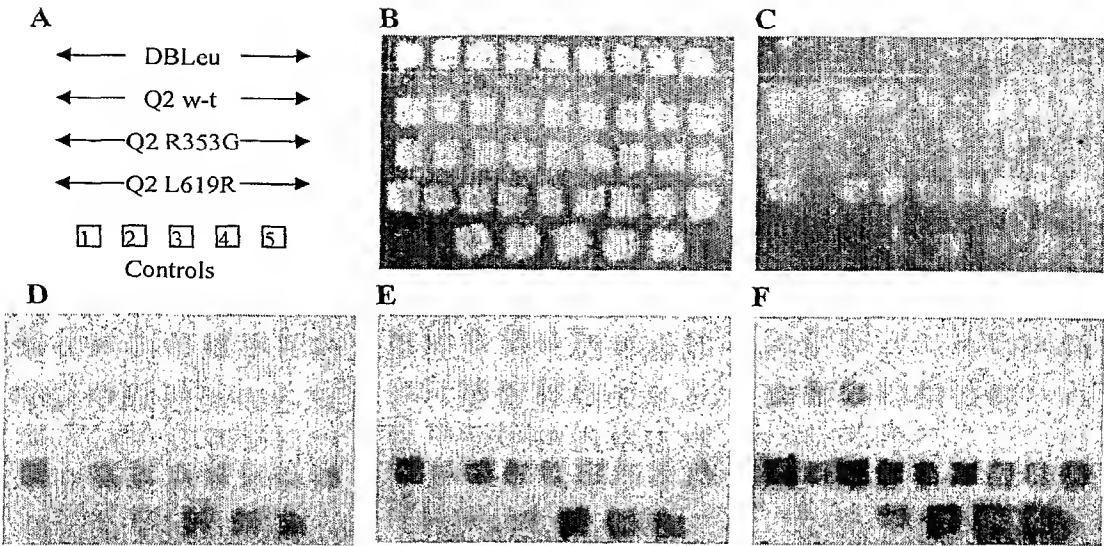


FIG. 4

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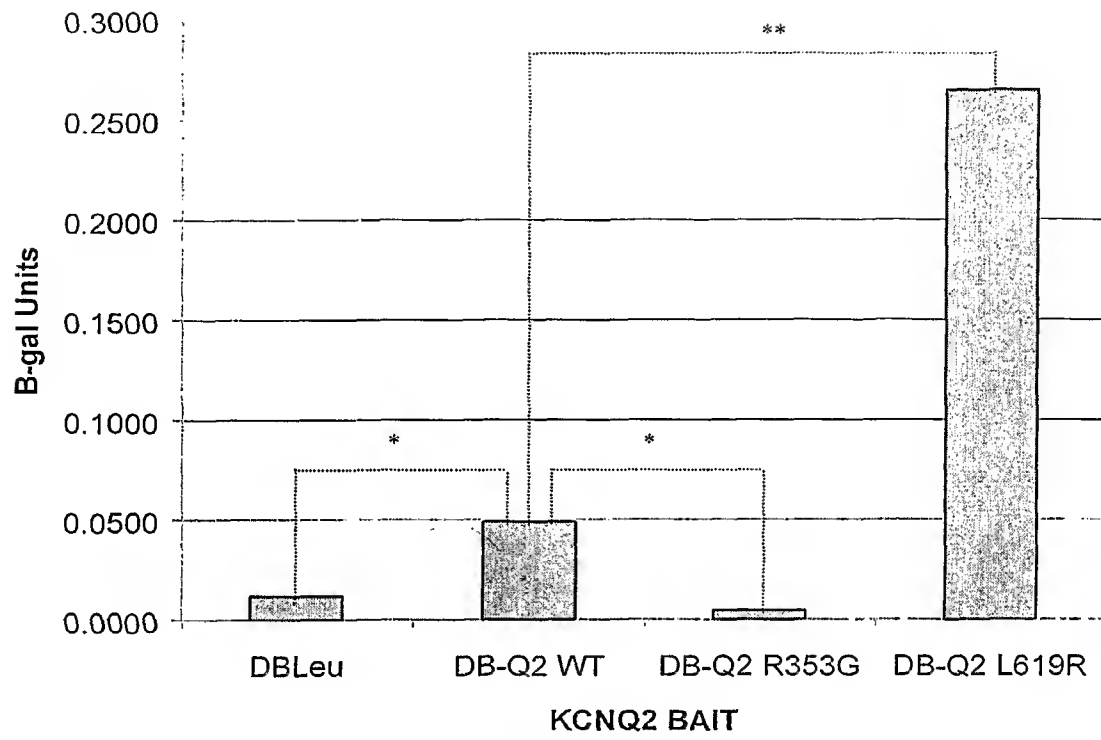


FIG. 5

SSCP Update Sequences.ST25  
SEQUENCE LISTING

&lt;110&gt; Bionomics Limited

&lt;120&gt; P15

&lt;130&gt; SSCP Update PCT

&lt;160&gt; 95

&lt;170&gt; PatentIn version 3.2

&lt;210&gt; 1

&lt;211&gt; 8381

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences .ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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385	

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ctgtttattg	gtgtcatcat agataatttc
120	
aaccagcaga	aaaagaagat aagtatttct
aatattttct	ctccactga aatagaaaat
180	



## SSCP Update Sequences.ST25

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 <212> DNA  
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 aatttttgggg aaaagaaaa tgatatgact tttcttacag gccacgttta agggatggat 120  
 ggatattatg tatgcagctg ttgattcacg aaat 154

<210> 23  
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 <212> DNA  
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 agggaaagca atctctcgat tcagtgccac ccctgccctt tacattttta ctcccttcaa 180  
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## SSCP update Sequences.ST25

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 gggccttttt gtccttattt cgtctcatga ctcaagactt ctgggaaaac ctttatcaac 240  
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<210> 25  
 <211> 388  
 <212> DNA  
 <213> Homo sapiens  
  
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 aaaaacagaa gaaagaaaaa gaaacagaaa gaacagtctg gagaagaaga gaaaaatgac 180  
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 gaaggaagta ggctgacata tgaaaagaga ttttcttctc cacaccaggt aaaaatatta 300  
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 <212> DNA  
 <213> Homo sapiens  
  
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 tgtcagccag gccagccgtg cctccagggt gctcccatc ctgcccata atgggaagat 360  
 gcatagcgct gtggactgca atggtgtggt ctccctgggtc gggggccctt ctaccctcac 420  
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<210> 27  
 <211> 221  
 <212> DNA  
 <213> Homo sapiens

<400> 27

## SSCP Update Sequences.ST25

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 atctgaaggc agcacggttg atattggagc tcccgccgag ggagaacagc ctgagggttg 180  
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 <213> Homo sapiens

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 <212> DNA  
 <213> Homo sapiens

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## SSCP Update Sequences.ST25

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 <212> DNA  
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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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&lt;400&gt; 34

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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&lt;212&gt; DNA

&lt;213&gt; Homo sapiens

&lt;400&gt; 36

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences .ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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&lt;400&gt; 54

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&lt;212&gt; DNA

&lt;213&gt; Homo sapiens

&lt;400&gt; 55

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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2189

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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<400> 61  
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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

&lt;213&gt; Homo sapiens

&lt;400&gt; 62

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ccagttgtac agcctgatgt aggacttgga aaacacatca atccaggaca aaagtgacgc   1860
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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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## SSCP Update Sequences.ST25

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<400> 73

## SSCP Update Sequences.ST25

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 50 55 60  
 Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80  
 Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95  
 Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105  
 Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140  
 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
 165 170 175  
 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
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 210 215 220  
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## SSCP Update Sequences.ST25

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 50 55 60  
 Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80  
 Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95  
 Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110  
 Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
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 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
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 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
 195 200 205  
 Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
 210 215 220  
 Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285

## SSCP Update Sequences.ST25

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Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
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Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335

Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350

Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365

Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe  
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<400> 75

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Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
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Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
 35 40 45

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
 50 55 60

Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80

Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95

Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110

Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125

Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140

Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 Page 124

145 150 155 160

Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
Page 125

SSCP Update Sequences.ST25  
425 430

420

Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
435 440 445

Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
450 455 460

Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
465 470 475 480

Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
485 490 495

Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
500 505 510

Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
515 520 525

Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
530 535 540

Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
545 550 555 560

Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
565 570 575

Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
580 585 590

Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
595 600 605

Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
610 615 620

Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
625 630 635 640

Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
645 650 655

Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
660 665 670

Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
675 680 685

Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
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## SSCP Update Sequences.ST25

690

695

700

Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720

Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735

Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750

Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765

Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780

Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800

Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815

Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830

Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845

Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860

Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880

Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895

Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910

Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925

Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940

Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960

Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met

## SSCP Update Sequences.ST25

965

970

975

Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
                   980                                  985                                  990

Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
           995                                  1000                                  1005

Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
       1010                                  1015                                  1020

Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
       1025                                  1030                                  1035

Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
       1040                                  1045                                  1050

Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
       1055                                  1060                                  1065

Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
       1070                                  1075                                  1080

Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
       1085                                  1090                                  1095

Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
       1100                                  1105                                  1110

Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
       1115                                  1120                                  1125

Thr Glu Asp Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
       1130                                  1135                                  1140

Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
       1145                                  1150                                  1155

Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
       1160                                  1165                                  1170

Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
       1175                                  1180                                  1185

Phe Lys Cys Cys Gln Ile Asn Val Glu Glu Gly Arg Gly Lys Gln  
       1190                                  1195                                  1200

Trp Trp Asn Leu Arg Arg Thr Cys Phe Arg Ile Val Glu His Asn  
       1205                                  1210                                  1215

Trp Phe Glu Thr Phe Ile Val Phe Met Ile Leu Leu Ser Ser Gly

1220 1225 1230

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## SSCP Update Sequences.ST25

1475													
Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile	Phe	Met	Thr	Glu
1490						1495					1500		
Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys	Leu	Gly	Ser	Lys
1505						1510					1515		
Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Gly	Asn	Lys	Phe	Gln	Gly
1520						1525					1530		
Val	Phe	Asp	Phe	Val	Thr	Arg	Gln	Val	Phe	Asp	Ile	Ser	Ile
1535						1540					1545		
Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met	Met	Val	Glu	Thr
1550						1555					1560		
Asp	Gln	Ser	Glu	Tyr	Val	Thr	Thr	Ile	Leu	Ser	Arg	Ile	Asn
1565						1570					1575		
Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys	Val	Leu	Lys	Leu
1580						1585					1590		
Ser	Leu	Arg	His	Tyr	Tyr	Phe	Thr	Ile	Gly	Trp	Asn	Ile	Phe
1595						1600					1605		
Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly	Met	Phe	Leu	Ala
1610						1615					1620		
Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr	Leu	Phe	Arg	Val
1625						1630					1635		
Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg	Leu	Ile	Lys	Gly
1640						1645					1650		
Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu	Met	Met	Ser	Leu
1655						1660					1665		
Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe	Leu	Val	Met	Phe
1670						1675					1680		
Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala	Tyr	Val	Lys	Arg
1685						1690					1695		
Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu	Thr	Phe	Gly	Asn
1700						1705					1710		
Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser	Ala	Gly	Trp	Asp
1715						1720					1725		
Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Lys	Pro	Pro	Asp	Cys	Asp

## SSCP Update Sequences.ST25

1730														
Asn	Lys	Val	Asn	Pro	Gly	Ser	Ser	Val	Lys	Gly	Asp	Cys	Gly	Asn
	1745					1750					1755			
Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser	Tyr	Ile	Ile	Ile	Ser
	1760					1765					1770			
Phe	Leu	Val	Val	Val	Asn	Met	Tyr	Ile	Ala	Val	Ile	Leu	Glu	Asn
	1775					1780					1785			
Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu	Pro	Leu	Ser	Glu	Asp
	1790					1795					1800			
Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu	Lys	Phe	Asp	Pro	Asp
	1805					1810					1815			
Ala	Thr	Gln	Phe	Met	Glu	Phe	Glu	Lys	Leu	Ser	Gln	Phe	Ala	Ala
	1820					1825					1830			
Ala	Leu	Glu	Pro	Pro	Leu	Asn	Leu	Pro	Gln	Pro	Asn	Lys	Leu	Gln
	1835					1840					1845			
Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser	Gly	Asp	Arg	Ile	His
	1850					1855					1860			
Cys	Leu	Asp	Ile	Leu	Phe	Ala	Phe	Thr	Lys	Arg	Val	Leu	Gly	Glu
	1865					1870					1875			
Ser	Gly	Glu	Met	Asp	Ala	Leu	Arg	Ile	Gln	Met	Glu	Glu	Arg	Phe
	1880					1885					1890			
Met	Ala	Ser	Asn	Pro	Ser	Lys	Val	Ser	Tyr	Gln	Pro	Ile	Thr	Thr
	1895					1900					1905			
Thr	Leu	Lys	Arg	Lys	Gln	Glu	Glu	Val	Ser	Ala	Val	Ile	Ile	Gln
	1910					1915					1920			
Arg	Ala	Tyr	Arg	Arg	His	Leu	Leu	Lys	Arg	Thr	Val	Lys	Gln	Ala
	1925					1930					1935			
Ser	Phe	Thr	Tyr	Asn	Lys	Asn	Lys	Ile	Lys	Gly	Gly	Ala	Asn	Leu
	1940					1945					1950			
Leu	Ile	Lys	Glu	Asp	Met	Ile	Ile	Asp	Arg	Ile	Asn	Glu	Asn	Ser
	1955					1960					1965			
Ile	Thr	Glu	Lys	Thr	Asp	Leu	Thr	Met	Ser	Thr	Ala	Ala	Cys	Pro
	1970					1975					1980			
Pro	Ser	Tyr	Asp	Arg	Val	Thr	Lys	Pro	Ile	Val	Glu	Lys	His	Glu

1985 SSCP Update Sequences.ST25  
1990 1995

Gln Glu Gly Lys Asp Glu Lys Ala Lys Gly Lys  
2000 2005

<210> 76  
<211> 2009  
<212> PRT  
<213> Homo sapiens  
<400> 76

Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
20 25 30

Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
35 40 45

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
50 55 60

Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
65 70 75 80

Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
85 90 95

Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
100 105 110

Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
115 120 125

Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
130 135 140

Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
145 150 155 160

Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
165 170 175

Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
180 185 190

Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
195 200 205

Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
210 215 220

## SSCP Update Sequences .ST25

Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285  
 Glu His Ser Ile Glu Lys Asn Ile Thr Val Asn Tyr Asn Gly Thr Leu  
 290 295 300  
 Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
 305 310 315 320  
 Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335  
 Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350  
 Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365  
 Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe Trp  
 370 375 380  
 Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr Tyr Met  
 385 390 395 400  
 Ile Phe Leu Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu Ile Asn  
 405 410 415  
 Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
 420 425 430  
 Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
 435 440 445  
 Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
 450 455 460  
 Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
 465 470 475 480  
 Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
 485 490 495

## SSCP Update Sequences.ST25

Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
 500 505 510  
 Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
 515 520 525  
 Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
 530 535 540  
 Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
 545 550 555 560  
 Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
 565 570 575  
 Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
 580 585 590  
 Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
 595 600 605  
 Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
 610 615 620  
 Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
 625 630 635 640  
 Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
 645 650 655  
 Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
 660 665 670  
 Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
 675 680 685  
 Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
 690 695 700  
 Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720  
 Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735  
 Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750  
 Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765



## SSCP Update Sequences.ST25

Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780  
 Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800  
 Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815  
 Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830  
 Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845  
 Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860  
 Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880  
 Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895  
 Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905  
 Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925  
 Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940  
 Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960  
 Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
 965 970 975  
 Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
 980 985 990  
 Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
 995 1000 1005  
 Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
 1010 1015 1020  
 Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
 1025 1030 1035

## SSCP Update Sequences.ST25

Ile	Arg	Lys	Gln	Lys	Ile	Leu	Asp	Glu	Ile	Lys	Pro	Leu	Asp	Asp
	1040					1045					1050			
Leu	Asn	Asn	Lys	Lys	Asp	Ser	Cys	Met	Ser	Asn	His	Thr	Thr	Glu
	1055					1060					1065			
Ile	Gly	Lys	Asp	Leu	Asp	Tyr	Leu	Lys	Asp	Val	Asn	Gly	Thr	Thr
	1070					1075					1080			
Ser	Gly	Ile	Gly	Thr	Gly	Ser	Ser	Val	Glu	Lys	Tyr	Ile	Ile	Asp
	1085					1090					1095			
Glu	Ser	Asp	Tyr	Met	Ser	Phe	Ile	Asn	Asn	Pro	Ser	Leu	Thr	Val
	1100					1105					1110			
Thr	Val	Pro	Ile	Ala	Val	Gly	Glu	Ser	Asp	Phe	Glu	Asn	Leu	Asn
	1115					1120					1125			
Thr	Glu	Asp	Phe	Ser	Ser	Glu	Ser	Asp	Leu	Glu	Glu	Ser	Lys	Glu
	1130					1135					1140			
Lys	Leu	Asn	Glu	Ser	Ser	Ser	Ser	Ser	Glu	Gly	Ser	Thr	Val	Asp
	1145					1150					1155			
Ile	Gly	Ala	Pro	Val	Glu	Glu	Gln	Pro	Val	Val	Glu	Pro	Glu	Glu
	1160					1165					1170			
Thr	Leu	Glu	Pro	Glu	Ala	Cys	Phe	Thr	Glu	Gly	Cys	Val	Gln	Arg
	1175					1180					1185			
Phe	Lys	Cys	Cys	Gln	Ile	Asn	Val	Glu	Glu	Gly	Arg	Gly	Lys	Gln
	1190					1195					1200			
Trp	Trp	Asn	Leu	Arg	Arg	Thr	Cys	Phe	Arg	Ile	Val	Glu	His	Asn
	1205					1210					1215			
Trp	Phe	Glu	Thr	Phe	Ile	Val	Phe	Met	Ile	Leu	Leu	Ser	Ser	Gly
	1220					1225					1230			
Ala	Leu	Ala	Phe	Glu	Asp	Ile	Tyr	Ile	Asp	Gln	Arg	Lys	Thr	Ile
	1235					1240					1245			
Lys	Thr	Met	Leu	Glu	Tyr	Ala	Asp	Lys	Val	Phe	Thr	Tyr	Ile	Phe
	1250					1255					1260			
Ile	Leu	Glu	Met	Leu	Leu	Lys	Trp	Val	Ala	Tyr	Gly	Tyr	Gln	Thr
	1265					1270					1275			
Tyr	Phe	Thr	Asn	Ala	Trp	Cys	Trp	Leu	Asp	Phe	Leu	Ile	Val	Asp
	1280					1285					1290			

## SSCP Update Sequences.ST25

Val	Ser	Leu	Val	Ser	Leu	Thr	Ala	Asn	Ala	Leu	Gly	Tyr	Ser	Glu
	1295					1300					1305			
Leu	Gly	Ala	Ile	Lys	Ser	Leu	Arg	Thr	Leu	Arg	Ala	Leu	Arg	Pro
	1310					1315					1320			
Leu	Arg	Ala	Leu	Ser	Arg	Phe	Glu	Gly	Met	Arg	Val	Val	Val	Asn
	1325					1330					1335			
Ala	Leu	Leu	Gly	Ala	Ile	Pro	Ser	Ile	Met	Asn	Val	Leu	Leu	Val
	1340					1345					1350			
Cys	Leu	Ile	Phe	Trp	Leu	Ile	Phe	Ser	Ile	Met	Gly	Val	Asn	Leu
	1355					1360					1365			
Phe	Ala	Gly	Lys	Phe	Tyr	His	Cys	Ile	Asn	Thr	Thr	Thr	Gly	Asp
	1370					1375					1380			
Arg	Phe	Asp	Ile	Glu	Asp	Val	Asn	Asn	His	Thr	Asp	Cys	Leu	Lys
	1385					1390					1395			
Leu	Ile	Glu	Arg	Asn	Glu	Thr	Ala	Arg	Trp	Lys	Asn	Val	Lys	Val
	1400					1405					1410			
Asn	Phe	Asp	Asn	Val	Gly	Phe	Gly	Tyr	Leu	Ser	Leu	Leu	Gln	Val
	1415					1420					1425			
Ala	Thr	Phe	Lys	Gly	Trp	Met	Asp	Ile	Met	Tyr	Ala	Ala	Val	Asp
	1430					1435					1440			
Ser	Arg	Asn	Val	Glu	Leu	Gln	Pro	Lys	Tyr	Glu	Lys	Ser	Leu	Tyr
	1445					1450					1455			
Met	Tyr	Leu	Tyr	Phe	Val	Ile	Phe	Ile	Ile	Phe	Gly	Ser	Phe	Phe
	1460					1465					1470			
Thr	Leu	Asn	Leu	Phe	Ile	Gly	Val	Ile	Ile	Asp	Asn	Phe	Asn	Gln
	1475					1480					1485			
Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile	Phe	Met	Thr	Glu	Glu
	1490					1495					1500			
Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys	Leu	Gly	Ser	Lys	Lys
	1505					1510					1515			
Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Gly	Asn	Lys	Phe	Gln	Gly	Met
	1520					1525					1530			
Val	Phe	Asp	Phe	Val	Thr	Arg	Gln	Val	Phe	Asp	Ile	Ser	Ile	Met
	1535					1540					1545			

## SSCP Update Sequences.ST25

Ile Leu Ile Cys Leu Asn Met Val Thr Met Met Val Glu Thr Asp  
 1550 1555 1560  
 Asp Gln Ser Glu Tyr Val Thr Thr Ile Leu Ser Arg Ile Asn Leu  
 1565 1570 1575  
 Val Phe Ile Val Leu Phe Thr Gly Glu Cys Val Leu Lys Leu Ile  
 1580 1585 1590  
 Ser Leu Arg His Tyr Tyr Phe Thr Ile Gly Trp Asn Ile Phe Asp  
 1595 1600 1605  
 Phe Val Val Val Ile Leu Ser Ile Val Gly Met Phe Leu Ala Glu  
 1610 1615 1620  
 Leu Ile Glu Lys Tyr Phe Val Ser Pro Thr Leu Phe Arg Val Ile  
 1625 1630 1635  
 Arg Leu Ala Arg Ile Gly Arg Ile Leu Arg Leu Ile Lys Gly Ala  
 1640 1645 1650  
 Lys Gly Ile Arg Thr Leu Leu Phe Ala Leu Met Met Ser Leu Pro  
 1655 1660 1665  
 Ala Leu Phe Asn Ile Gly Leu Leu Leu Phe Leu Val Met Phe Ile  
 1670 1675 1680  
 Tyr Ala Ile Phe Gly Met Ser Asn Phe Ala Tyr Val Lys Arg Glu  
 1685 1690 1695  
 Val Gly Ile Asp Asp Met Phe Asn Phe Glu Thr Phe Gly Asn Ser  
 1700 1705 1710  
 Met Ile Cys Leu Phe Gln Ile Thr Thr Ser Ala Gly Trp Asp Gly  
 1715 1720 1725  
 Leu Leu Ala Pro Ile Leu Asn Ser Lys Pro Pro Asp Cys Asp Pro  
 1730 1735 1740  
 Asn Lys Val Asn Pro Gly Ser Ser Val Lys Gly Asp Cys Gly Asn  
 1745 1750 1755  
 Pro Ser Val Gly Ile Phe Phe Phe Val Ser Tyr Ile Ile Ile Ser  
 1760 1765 1770  
 Phe Leu Val Val Val Asn Met Tyr Ile Ala Val Ile Leu Glu Asn  
 1775 1780 1785  
 Phe Ser Val Ala Thr Glu Glu Ser Ala Glu Pro Leu Ser Glu Asp  
 1790 1795 1800

## SSCP Update Sequences.ST25

Asp Phe Glu Met Phe Tyr Glu Val Trp Glu Lys Phe Asp Pro Asp  
 1805 1810 1815  
 Ala Thr Gln Phe Met Glu Phe Glu Lys Leu Ser Gln Phe Ala Ala  
 1820 1825 1830  
 Ala Leu Glu Pro Pro Leu Asn Leu Pro Gln Pro Asn Lys Leu Gln  
 1835 1840 1845  
 Leu Ile Ala Met Asp Leu Pro Met Val Ser Gly Asp Arg Ile His  
 1850 1855 1860  
 Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys Arg Val Leu Gly Glu  
 1865 1870 1875  
 Ser Gly Glu Met Asp Ala Leu Arg Ile Gln Met Glu Glu Arg Phe  
 1880 1885 1890  
 Met Ala Ser Asn Pro Ser Lys Val Ser Tyr Gln Pro Ile Thr Thr  
 1895 1900 1905  
 Thr Leu Lys Arg Lys Gln Glu Glu Val Ser Ala Val Ile Ile Gln  
 1910 1915 1920  
 Arg Ala Tyr Arg Arg His Leu Leu Lys Arg Thr Val Lys Gln Ala  
 1925 1930 1935  
 Ser Phe Thr Tyr Asn Lys Asn Lys Ile Lys Gly Gly Ala Asn Leu  
 1940 1945 1950  
 Leu Ile Lys Glu Asp Met Ile Ile Asp Arg Ile Asn Glu Asn Ser  
 1955 1960 1965  
 Ile Thr Glu Lys Thr Asp Leu Thr Met Ser Thr Ala Ala Cys Pro  
 1970 1975 1980  
 Pro Ser Tyr Asp Arg Val Thr Lys Pro Ile Val Glu Lys His Glu  
 1985 1990 1995  
 Gln Glu Gly Lys Asp Glu Lys Ala Lys Gly Lys  
 2000 2005  
 <210> 77  
 <211> 2009  
 <212> PRT  
 <213> Homo sapiens  
 <400> 77  
 Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
 1 5 10 15

## SSCP Update Sequences.ST25

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
 20 25 30  
 Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
 35 40 45  
 Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
 50 55 60  
 Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80  
 Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95  
 Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110  
 Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140  
 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
 165 170 175  
 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
 195 200 205  
 Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
 210 215 220  
 Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285

## SSCP Update Sequences.ST25

Glu His Ser Ile Glu Lys Asn Ile Thr Val Asn Tyr Asn Gly Thr Leu  
 290 295 300  
 Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
 305 310 315 320  
 Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335  
 Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350  
 Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365  
 Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe Trp  
 370 375 380  
 Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr Tyr Met  
 385 390 395 400  
 Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Asn Leu Ile Asn  
 405 410 415  
 Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
 420 425 430  
 Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
 435 440 445  
 Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
 450 455 460  
 Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
 465 470 475 480  
 Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
 485 490 495  
 Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
 500 505 510  
 Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
 515 520 525  
 Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
 530 535 540  
 Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
 545 550 555 560

## SSCP Update Sequences.ST25

Gly Ser Leu Phe Ser<sub>565</sub> Pro Arg Arg Asn Ser<sub>570</sub> Arg Thr Ser Leu Phe Ser<sub>575</sub>  
 Phe Arg Gly Arg<sub>580</sub> Ala Lys Asp Val Gly<sub>585</sub> Ser Glu Asn Asp Phe<sub>590</sub> Ala Asp  
 Asp Glu His<sub>595</sub> Ser Thr Phe Glu Asp<sub>600</sub> Asn Glu Ser Arg Arg<sub>605</sub> Asp Ser Leu  
 Phe Val<sub>610</sub> Pro Arg Arg His Gly<sub>615</sub> Glu Arg Arg Asn Ser<sub>620</sub> Asn Leu Ser Gln  
 Thr Ser Arg Ser Ser Arg<sub>630</sub> Met Leu Ala Val Phe<sub>635</sub> Pro Ala Asn Gly Lys<sub>640</sub>  
 Met His Ser Thr Val<sub>645</sub> Asp Cys Asn Gly Val<sub>650</sub> Val Ser Leu Val Gly<sub>655</sub> Gly  
 Pro Ser Val Pro<sub>660</sub> Thr Ser Pro Val Gly<sub>665</sub> Gln Leu Leu Pro Glu<sub>670</sub> Val Ile  
 Ile Asp Lys<sub>675</sub> Pro Ala Thr Asp Asp<sub>680</sub> Asn Gly Thr Thr Thr<sub>685</sub> Glu Thr Glu  
 Met Arg<sub>690</sub> Lys Arg Arg Ser Ser<sub>695</sub> Ser Phe His Val Ser<sub>700</sub> Met Asp Phe Leu  
 Glu Asp Pro Ser Gln Arg<sub>710</sub> Gln Arg Ala Met Ser<sub>715</sub> Ile Ala Ser Ile Leu<sub>720</sub>  
 Thr Asn Thr Val Glu<sub>725</sub> Glu Leu Glu Glu Ser<sub>730</sub> Arg Gln Lys Cys Pro<sub>735</sub> Pro  
 Cys Trp Tyr Lys<sub>740</sub> Phe Ser Asn Ile Phe<sub>745</sub> Leu Ile Trp Asp Cys<sub>750</sub> Ser Pro  
 Tyr Trp Leu<sub>755</sub> Lys Val Lys His Val<sub>760</sub> Val Asn Leu Val Val<sub>765</sub> Met Asp Pro  
 Phe Val<sub>770</sub> Asp Leu Ala Ile Thr<sub>775</sub> Ile Cys Ile Val Leu<sub>780</sub> Asn Thr Leu Phe  
 Met Ala Met Glu His Tyr<sub>790</sub> Pro Met Thr Asp His<sub>795</sub> Phe Asn Asn Val Leu<sub>800</sub>  
 Thr Val Gly Asn Leu<sub>805</sub> Val Phe Thr Gly Ile<sub>810</sub> Phe Thr Ala Glu Met<sub>815</sub> Phe  
 Leu Lys Ile Ile<sub>820</sub> Ala Met Asp Pro Tyr<sub>825</sub> Tyr Tyr Phe Gln Glu<sub>830</sub> Gly Trp



## SSCP Update Sequences.ST25

Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845  
 Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860  
 Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880  
 Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895  
 Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910  
 Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925  
 Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940  
 Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960  
 Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
 965 970 975  
 Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
 980 985 990  
 Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
 995 1000 1005  
 Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
 1010 1015 1020  
 Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
 1025 1030 1035  
 Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
 1040 1045 1050  
 Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
 1055 1060 1065  
 Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
 1070 1075 1080  
 Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
 1085 1090 1095

## SSCP Update Sequences.ST25

Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
 1100 1105 1110  
 Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
 1115 1120 1125  
 Thr Glu Asp, Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
 1130 1135 1140  
 Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
 1145 1150 1155  
 Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
 1160 1165 1170  
 Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
 1175 1180 1185  
 Phe Lys Cys Cys Gln Ile Asn Val Glu Glu Gly Arg Gly Lys Gln  
 1190 1195 1200  
 Trp Trp Asn Leu Arg Arg Thr Cys Phe Arg Ile Val Glu His Asn  
 1205 1210 1215  
 Trp Phe Glu Thr Phe Ile Val Phe Met Ile Leu Leu Ser Ser Gly  
 1220 1225 1230  
 Ala Leu Ala Phe Glu Asp Ile Tyr Ile Asp Gln Arg Lys Thr Ile  
 1235 1240 1245  
 Lys Thr Met Leu Glu Tyr Ala Asp Lys Val Phe Thr Tyr Ile Phe  
 1250 1255 1260  
 Ile Leu Glu Met Leu Leu Lys Trp Val Ala Tyr Gly Tyr Gln Thr  
 1265 1270 1275  
 Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp Phe Leu Ile Val Asp  
 1280 1285 1290  
 Val Ser Leu Val Ser Leu Thr Ala Asn Ala Leu Gly Tyr Ser Glu  
 1295 1300 1305  
 Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu Arg Ala Leu Arg Pro  
 1310 1315 1320  
 Leu Arg Ala Leu Ser Arg Phe Glu Gly Met Arg Val Val Val Asn  
 1325 1330 1335  
 Ala Leu Leu Gly Ala Ile Pro Ser Ile Met Asn Val Leu Leu Val  
 1340 1345 1350

## SSCP Update Sequences.ST25

Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile Met Gly Val Asn Leu  
 1355 1360 1365  
 Phe Ala Gly Lys Phe Tyr His Cys Ile Asn Thr Thr Thr Gly Asp  
 1370 1375 1380  
 Arg Phe Asp Ile Glu Asp Val Asn Asn His Thr Asp Cys Leu Lys  
 1385 1390 1395  
 Leu Ile Glu Arg Asn Glu Thr Ala Arg Trp Lys Asn Val Lys Val  
 1400 1405 1410  
 Asn Phe Asp Asn Val Gly Phe Gly Tyr Leu Ser Leu Leu Gln Val  
 1415 1420 1425  
 Ala Thr Phe Lys Gly Trp Met Asp Ile Met Tyr Ala Ala Val Asp  
 1430 1435 1440  
 Ser Arg Asn Val Glu Leu Gln Pro Lys Tyr Glu Lys Ser Leu Tyr  
 1445 1450 1455  
 Met Tyr Leu Tyr Phe Val Ile Phe Ile Ile Phe Gly Ser Phe Phe  
 1460 1465 1470  
 Thr Leu Asn Leu Phe Ile Gly Val Ile Ile Asp Asn Phe Asn Gln  
 1475 1480 1485  
 Gln Lys Lys Lys Phe Gly Gly Gln Asp Ile Phe Met Thr Glu Glu  
 1490 1495 1500  
 Gln Lys Lys Tyr Tyr Asn Ala Met Lys Lys Leu Gly Ser Lys Lys  
 1505 1510 1515  
 Pro Gln Lys Pro Ile Pro Arg Pro Gly Asn Lys Phe Gln Gly Met  
 1520 1525 1530  
 Val Phe Asp Phe Val Thr Arg Gln Val Phe Asp Ile Ser Ile Met  
 1535 1540 1545  
 Ile Leu Ile Cys Leu Asn Met Val Thr Met Met Val Glu Thr Asp  
 1550 1555 1560  
 Asp Gln Ser Glu Tyr Val Thr Thr Ile Leu Ser Arg Ile Asn Leu  
 1565 1570 1575  
 Val Phe Ile Val Leu Phe Thr Gly Glu Cys Val Leu Lys Leu Ile  
 1580 1585 1590  
 Ser Leu Arg His Tyr Tyr Phe Thr Ile Gly Trp Asn Ile Phe Asp  
 1595 1600 1605

## SSCP Update Sequences.ST25

Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly	Met	Phe	Leu	Ala	Glu
1610						1615					1620			
Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr	Leu	Phe	Arg	Val	Ile
1625						1630					1635			
Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg	Leu	Ile	Lys	Gly	Ala
1640						1645					1650			
Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu	Met	Met	Ser	Leu	Pro
1655						1660					1665			
Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe	Leu	Val	Met	Phe	Ile
1670						1675					1680			
Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala	Tyr	Val	Lys	Arg	Glu
1685						1690					1695			
Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu	Thr	Phe	Gly	Asn	Ser
1700						1705					1710			
Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser	Ala	Gly	Trp	Asp	Gly
1715						1720					1725			
Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Lys	Pro	Pro	Asp	Cys	Asp	Pro
1730						1735					1740			
Asn	Lys	Val	Asn	Pro	Gly	Ser	Ser	Val	Lys	Gly	Asp	Cys	Gly	Asn
1745						1750					1755			
Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser	Tyr	Ile	Ile	Ile	Ser
1760						1765					1770			
Phe	Leu	Val	Val	Val	Asn	Met	Tyr	Ile	Ala	Val	Ile	Leu	Glu	Asn
1775						1780					1785			
Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu	Pro	Leu	Ser	Glu	Asp
1790						1795					1800			
Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu	Lys	Phe	Asp	Pro	Asp
1805						1810					1815			
Ala	Thr	Gln	Phe	Met	Glu	Phe	Glu	Lys	Leu	Ser	Gln	Phe	Ala	Ala
1820						1825					1830			
Ala	Leu	Glu	Pro	Pro	Leu	Asn	Leu	Pro	Gln	Pro	Asn	Lys	Leu	Gln
1835						1840					1845			
Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser	Gly	Asp	Arg	Ile	His
1850						1855					1860			

## SSCP Update Sequences.ST25

Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys Arg Val Leu Gly Glu  
1865 1870 1875

Ser Gly Glu Met Asp Ala Leu Arg Ile Gln Met Glu Glu Arg Phe  
1880 1885 1890

Met Ala Ser Asn Pro Ser Lys Val Ser Tyr Gln Pro Ile Thr Thr  
1895 1900 1905

Thr Leu Lys Arg Lys Gln Glu Glu Val Ser Ala Val Ile Ile Gln  
1910 1915 1920

Arg Ala Tyr Arg Arg His Leu Leu Lys Arg Thr Val Lys Gln Ala  
1925 1930 1935

Ser Phe Thr Tyr Asn Lys Asn Lys Ile Lys Gly Gly Ala Asn Leu  
1940 1945 1950

Leu Ile Lys Glu Asp Met Ile Ile Asp Arg Ile Asn Glu Asn Ser  
1955 1960 1965

Ile Thr Glu Lys Thr Asp Leu Thr Met Ser Thr Ala Ala Cys Pro  
1970 1975 1980

Pro Ser Tyr Asp Arg Val Thr Lys Pro Ile Val Glu Lys His Glu  
1985 1990 1995

Gln Glu Gly Lys Asp Glu Lys Ala Lys Gly Lys  
2000 2005

<210> 78  
<211> 2009  
<212> PRT  
<213> Homo sapiens

<400> 78

Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
20 25 30

Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
35 40 45

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
50 55 60

Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
65 70 75 80

## SSCP Update Sequences.ST25

Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95  
 Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110  
 Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140  
 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
 165 170 175  
 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
 195 200 205  
 Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
 210 215 220  
 Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285  
 Glu His Ser Ile Glu Lys Asn Ile Thr Val Asn Tyr Asn Gly Thr Leu  
 290 295 300  
 Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
 305 310 315 320  
 Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335  
 Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350

## SSCP Update Sequences.ST25

Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365  
 Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe Trp  
 370 375 380  
 Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr Tyr Met  
 385 390 395 400  
 Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu Ile Asn  
 405 410 415  
 Leu Ile Leu Ala Val Glu Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
 420 425 430  
 Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
 435 440 445  
 Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
 450 455 460  
 Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
 465 470 475 480  
 Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
 485 490 495  
 Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
 500 505 510  
 Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
 515 520 525  
 Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
 530 535 540  
 Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
 545 550 555 560  
 Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
 565 570 575  
 Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
 580 585 590  
 Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
 595 600 605  
 Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
 610 615 620

## SSCP Update Sequences.ST25

Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
 625 630 635 640  
 Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
 645 650 655  
 Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
 660 665 670  
 Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
 675 680 685  
 Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
 690 695 700  
 Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720  
 Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735  
 Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750  
 Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765  
 Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780  
 Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800  
 Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815  
 Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830  
 Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845  
 Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860  
 Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880  
 Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895



## SSCP Update Sequences.ST25

Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910

Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925

Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940

Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960

Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
 965 970 975

Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
 980 985 990

Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
 995 1000 1005

Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
 1010 1015 1020

Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
 1025 1030 1035

Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
 1040 1045 1050

Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
 1055 1060 1065

Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
 1070 1075 1080

Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
 1085 1090 1095

Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
 1100 1105 1110

Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
 1115 1120 1125

Thr Glu Asp Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
 1130 1135 1140

Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
 1145 1150 1155

## SSCP Update Sequences.ST25

Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
1160 1165 1170

Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
1175 1180 1185

Phe Lys Cys Cys Gln Ile Asn Val Glu Glu Gly Arg Gly Lys Gln  
1190 1195 1200

Trp Trp Asn Leu Arg Arg Thr Cys Phe Arg Ile Val Glu His Asn  
1205 1210 1215

Trp Phe Glu Thr Phe Ile Val Phe Met Ile Leu Leu Ser Ser Gly  
1220 1225 1230

Ala Leu Ala Phe Glu Asp Ile Tyr Ile Asp Gln Arg Lys Thr Ile  
1235 1240 1245

Lys Thr Met Leu Glu Tyr Ala Asp Lys Val Phe Thr Tyr Ile Phe  
1250 1255 1260

Ile Leu Glu Met Leu Leu Lys Trp Val Ala Tyr Gly Tyr Gln Thr  
1265 1270 1275

Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp Phe Leu Ile Val Asp  
1280 1285 1290

Val Ser Leu Val Ser Leu Thr Ala Asn Ala Leu Gly Tyr Ser Glu  
1295 1300 1305

Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu Arg Ala Leu Arg Pro  
1310 1315 1320

Leu Arg Ala Leu Ser Arg Phe Glu Gly Met Arg Val Val Val Asn  
1325 1330 1335

Ala Leu Leu Gly Ala Ile Pro Ser Ile Met Asn Val Leu Leu Val  
1340 1345 1350

Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile Met Gly Val Asn Leu  
1355 1360 1365

Phe Ala Gly Lys Phe Tyr His Cys Ile Asn Thr Thr Thr Gly Asp  
1370 1375 1380

Arg Phe Asp Ile Glu Asp Val Asn Asn His Thr Asp Cys Leu Lys  
1385 1390 1395

Leu Ile Glu Arg Asn Glu Thr Ala Arg Trp Lys Asn Val Lys Val  
1400 1405 1410

## SSCP Update Sequences.ST25

Asn	Phe	Asp	Asn	Val	Gly	Phe	Gly	Tyr	Leu	Ser	Leu	Leu	Gln	Val
1415						1420					1425			
Ala	Thr	Phe	Lys	Gly	Trp	Met	Asp	Ile	Met	Tyr	Ala	Ala	Val	Asp
1430						1435					1440			
Ser	Arg	Asn	Val	Glu	Leu	Gln	Pro	Lys	Tyr	Glu	Lys	Ser	Leu	Tyr
1445						1450					1455			
Met	Tyr	Leu	Tyr	Phe	Val	Ile	Phe	Ile	Ile	Phe	Gly	Ser	Phe	Phe
1460						1465					1470			
Thr	Leu	Asn	Leu	Phe	Ile	Gly	Val	Ile	Ile	Asp	Asn	Phe	Asn	Gln
1475						1480					1485			
Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile	Phe	Met	Thr	Glu	Glu
1490						1495					1500			
Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys	Leu	Gly	Ser	Lys	Lys
1505						1510					1515			
Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Gly	Asn	Lys	Phe	Gln	Gly	Met
1520						1525					1530			
Val	Phe	Asp	Phe	Val	Thr	Arg	Gln	Val	Phe	Asp	Ile	Ser	Ile	Met
1535						1540					1545			
Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met	Met	Val	Glu	Thr	Asp
1550						1555					1560			
Asp	Gln	Ser	Glu	Tyr	Val	Thr	Thr	Ile	Leu	Ser	Arg	Ile	Asn	Leu
1565						1570					1575			
Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys	Val	Leu	Lys	Leu	Ile
1580						1585					1590			
Ser	Leu	Arg	His	Tyr	Tyr	Phe	Thr	Ile	Gly	Trp	Asn	Ile	Phe	Asp
1595						1600					1605			
Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly	Met	Phe	Leu	Ala	Glu
1610						1615					1620			
Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr	Leu	Phe	Arg	Val	Ile
1625						1630					1635			
Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg	Leu	Ile	Lys	Gly	Ala
1640						1645					1650			
Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu	Met	Met	Ser	Leu	Pro
1655						1660					1665			

## SSCP Update Sequences.ST25

Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe	Leu	Val	Met	Phe	Ile
	1670					1675					1680			
Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala	Tyr	Val	Lys	Arg	Glu
	1685					1690					1695			
Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu	Thr	Phe	Gly	Asn	Ser
	1700					1705					1710			
Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser	Ala	Gly	Trp	Asp	Gly
	1715					1720					1725			
Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Lys	Pro	Pro	Asp	Cys	Asp	Pro
	1730					1735					1740			
Asn	Lys	Val	Asn	Pro	Gly	Ser	Ser	Val	Lys	Gly	Asp	Cys	Gly	Asn
	1745					1750					1755			
Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser	Tyr	Ile	Ile	Ile	Ser
	1760					1765					1770			
Phe	Leu	Val	Val	Val	Asn	Met	Tyr	Ile	Ala	Val	Ile	Leu	Glu	Asn
	1775					1780					1785			
Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu	Pro	Leu	Ser	Glu	Asp
	1790					1795					1800			
Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu	Lys	Phe	Asp	Pro	Asp
	1805					1810					1815			
Ala	Thr	Gln	Phe	Met	Glu	Phe	Glu	Lys	Leu	Ser	Gln	Phe	Ala	Ala
	1820					1825					1830			
Ala	Leu	Glu	Pro	Pro	Leu	Asn	Leu	Pro	Gln	Pro	Asn	Lys	Leu	Gln
	1835					1840					1845			
Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser	Gly	Asp	Arg	Ile	His
	1850					1855					1860			
Cys	Leu	Asp	Ile	Leu	Phe	Ala	Phe	Thr	Lys	Arg	Val	Leu	Gly	Glu
	1865					1870					1875			
Ser	Gly	Glu	Met	Asp	Ala	Leu	Arg	Ile	Gln	Met	Glu	Glu	Arg	Phe
	1880					1885					1890			
Met	Ala	Ser	Asn	Pro	Ser	Lys	Val	Ser	Tyr	Gln	Pro	Ile	Thr	Thr
	1895					1900					1905			
Thr	Leu	Lys	Arg	Lys	Gln	Glu	Glu	Val	Ser	Ala	Val	Ile	Ile	Gln
	1910					1915					1920			

## SSCP Update Sequences.ST25

Arg Ala Tyr Arg Arg His Leu Leu Lys Arg Thr Val Lys Gln Ala  
 1925 1930 1935

Ser Phe Thr Tyr Asn Lys Asn Lys Ile Lys Gly Gly Ala Asn Leu  
 1940 1945 1950

Leu Ile Lys Glu Asp Met Ile Ile Asp Arg Ile Asn Glu Asn Ser  
 1955 1960 1965

Ile Thr Glu Lys Thr Asp Leu Thr Met Ser Thr Ala Ala Cys Pro  
 1970 1975 1980

Pro Ser Tyr Asp Arg Val Thr Lys Pro Ile Val Glu Lys His Glu  
 1985 1990 1995

Gln Glu Gly Lys Asp Glu Lys Ala Lys Gly Lys  
 2000 2005

<210> 79  
 <211> 1406  
 <212> PRT  
 <213> Homo sapiens

<400> 79

Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
 1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
 20 25 30

Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
 35 40 45

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
 50 55 60

Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80

Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95

Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110

Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125

Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140

Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 Page 155

SSCP Update Sequences.ST25

145		150		155		160									
Phe	Thr	Gly	Ile	Tyr	Thr	Phe	Glu	Ser	Leu	Ile	Lys	Ile	Ile	Ala	Arg
				165					170					175	
Gly	Phe	Cys	Leu	Glu	Asp	Phe	Thr	Phe	Leu	Arg	Asp	Pro	Trp	Asn	Trp
			180					185					190		
Leu	Asp	Phe	Thr	Val	Ile	Thr	Phe	Ala	Tyr	Val	Thr	Glu	Phe	Val	Asp
		195					200					205			
Leu	Gly	Asn	Val	Ser	Ala	Leu	Arg	Thr	Phe	Arg	Val	Leu	Arg	Ala	Leu
	210					215					220				
Lys	Thr	Ile	Ser	Val	Ile	Pro	Gly	Leu	Lys	Thr	Ile	Val	Gly	Ala	Leu
225					230					235					240
Ile	Gln	Ser	Val	Lys	Lys	Leu	Ser	Asp	Val	Met	Ile	Leu	Thr	Val	Phe
				245					250					255	
Cys	Leu	Ser	Val	Phe	Ala	Leu	Ile	Gly	Leu	Gln	Leu	Phe	Met	Gly	Asn
			260					265					270		
Leu	Arg	Asn	Lys	Cys	Ile	Gln	Trp	Pro	Pro	Thr	Asn	Ala	Ser	Leu	Glu
		275					280					285			
Glu	His	Ser	Ile	Glu	Lys	Asn	Ile	Thr	Val	Asn	Tyr	Asn	Gly	Thr	Leu
	290					295					300				
Ile	Asn	Glu	Thr	Val	Phe	Glu	Phe	Asp	Trp	Lys	Ser	Tyr	Ile	Gln	Asp
305					310					315					320
Ser	Arg	Tyr	His	Tyr	Phe	Leu	Glu	Gly	Phe	Leu	Asp	Ala	Leu	Leu	Cys
				325					330					335	
Gly	Asn	Ser	Ser	Asp	Ala	Gly	Gln	Cys	Pro	Glu	Gly	Tyr	Met	Cys	Val
			340					345					350		
Lys	Ala	Gly	Arg	Asn	Pro	Asn	Tyr	Gly	Tyr	Thr	Ser	Phe	Asp	Thr	Phe
		355					360					365			
Ser	Trp	Ala	Phe	Leu	Ser	Leu	Phe	Arg	Leu	Met	Thr	Gln	Asp	Phe	Trp
	370					375					380				
Glu	Asn	Leu	Tyr	Gln	Leu	Thr	Leu	Arg	Ala	Ala	Gly	Lys	Thr	Tyr	Met
385					390					395					400
Ile	Phe	Phe	Val	Leu	Val	Ile	Phe	Leu	Gly	Ser	Phe	Tyr	Leu	Ile	Asn
				405					410					415	
Leu	Ile	Leu	Ala	Val	Val	Ala	Met	Ala	Tyr	Glu	Glu	Gln	Asn	Gln	Ala

SSCP Update Sequences.ST25  
425 430

420

Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
435 440 445

Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
450 455 460

Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
465 470 475 480

Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
485 490 495

Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
500 505 510

Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
515 520 525

Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
530 535 540

Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
545 550 555 560

Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
565 570 575

Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
580 585 590

Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
595 600 605

Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
610 615 620

Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
625 630 635 640

Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
645 650 655

Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
660 665 670

Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
675 680 685

Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
Page 157

## SSCP update Sequences.ST25

690

695

700

Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720

Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735

Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750

Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765

Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780

Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800

Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815

Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830

Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845

Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860

Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880

Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895

Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910

Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925

Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940

Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960

Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
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## SSCP update Sequences.ST25

965

970

975

Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
                   980                                  985                                  990

Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
                   995                                  1000                                  1005

Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
           1010                                  1015                                  1020

Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
           1025                                  1030                                  1035

Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
           1040                                  1045                                  1050

Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
           1055                                  1060                                  1065

Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
           1070                                  1075                                  1080

Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
           1085                                  1090                                  1095

Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
           1100                                  1105                                  1110

Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
           1115                                  1120                                  1125

Thr Glu Asp Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
           1130                                  1135                                  1140

Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
           1145                                  1150                                  1155

Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
           1160                                  1165                                  1170

Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
           1175                                  1180                                  1185

Phe Lys Cys Cys Gln Ile Asn Val Glu Glu Gly Arg Gly Lys Gln  
           1190                                  1195                                  1200

Trp Trp Asn Leu Arg Arg Thr Cys Phe Arg Ile Val Glu His Asn  
           1205                                  1210                                  1215

Trp Phe Glu Thr Phe Ile Val Phe Met Ile Leu Leu Ser Ser Gly

## SSCP Update Sequences.ST25

1220 1225 1230  
 Ala Leu Ala Phe Glu Asp Ile Tyr Ile Asp Gln Arg Lys Thr Ile  
 1235 1240 1245  
 Lys Thr Met Leu Glu Tyr Ala Asp Lys Val Phe Thr Tyr Ile Phe  
 1250 1255 1260  
 Ile Leu Glu Met Leu Leu Lys Trp Val Ala Tyr Gly Tyr Gln Thr  
 1265 1270 1275  
 Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp Phe Leu Ile Val Asp  
 1280 1285 1290  
 Val Ser Leu Val Ser Leu Thr Ala Asn Ala Leu Gly Tyr Ser Glu  
 1295 1300 1305  
 Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu Arg Ala Leu Arg Pro  
 1310 1315 1320  
 Leu Arg Ala Leu Ser Arg Phe Glu Gly Met Arg Val Val Val Asn  
 1325 1330 1335  
 Ala Leu Leu Gly Ala Ile Pro Ser Ile Met Asn Val Leu Leu Val  
 1340 1345 1350  
 Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile Met Gly Val Asn Leu  
 1355 1360 1365  
 Phe Ala Gly Lys Phe Tyr His Cys Ile Asn Thr Thr Thr Gly Asp  
 1370 1375 1380  
 Arg Phe Asp Ile Glu Asp Val Asn Asn His Thr Asp Cys Leu Lys  
 1385 1390 1395  
 Leu Ile Glu Arg Asn Glu Thr Ala  
 1400 1405

<210> 80  
 <211> 2009  
 <212> PRT  
 <213> Homo sapiens

<400> 80

Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
 1 5 10 15  
 Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
 20 25 30  
 Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
 35 40 45

## SSCP update Sequences.ST25

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
 50 55 60  
 Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
 65 70 75 80  
 Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
 85 90 95  
 Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
 100 105 110  
 Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140  
 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
 165 170 175  
 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
 195 200 205  
 Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
 210 215 220  
 Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285  
 Glu His Ser Ile Glu Lys Asn Ile Thr Val Asn Tyr Asn Gly Thr Leu  
 290 295 300  
 Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
 305 310 315 320

## SSCP Update Sequences .ST25

Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335  
 Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350  
 Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365  
 Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe Trp  
 370 375 380  
 Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr Tyr Met  
 385 390 395 400  
 Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu Ile Asn  
 405 410 415  
 Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
 420 425  
 Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
 435 440 445  
 Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
 450 455 460  
 Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
 465 470 475 480  
 Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
 485 490 495  
 Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
 500 505 510  
 Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
 515 520 525  
 Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
 530 535 540  
 Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
 545 550 555 560  
 Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
 565 570 575  
 Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
 580 585 590

## SSCP Update Sequences.ST25

Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
 595 600 605  
 Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
 610 615 620  
 Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
 625 630 635 640  
 Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
 645 650 655  
 Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
 660 665 670  
 Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
 675 680 685  
 Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
 690 695 700  
 Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720  
 Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735  
 Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750  
 Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765  
 Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780  
 Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800  
 Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815  
 Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830  
 Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845  
 Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860

## SSCP Update Sequences.ST25

Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880  
 Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895  
 Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910  
 Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925  
 Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940  
 Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960  
 Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
 965 970 975  
 Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
 980 985 990  
 Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
 995 1000 1005  
 Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
 1010 1015 1020  
 Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
 1025 1030 1035  
 Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
 1040 1045 1050  
 Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
 1055 1060 1065  
 Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
 1070 1075 1080  
 Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
 1085 1090 1095  
 Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
 1100 1105 1110  
 Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
 1115 1120 1125

## SSCP Update Sequences.ST25

Thr Glu Asp Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
 1130 1135 1140  
 Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
 1145 1150 1155  
 Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
 1160 1165 1170  
 Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
 1175 1180 1185  
 Phe Lys Cys Cys Gln Ile Asn Val Glu Glu Gly Arg Gly Lys Gln  
 1190 1195 1200  
 Trp Trp Asn Leu Arg Arg Thr Cys Phe Arg Ile Val Glu His Asn  
 1205 1210 1215  
 Trp Phe Glu Thr Phe Ile Val Phe Met Ile Leu Leu Ser Ser Gly  
 1220 1225 1230  
 Ala Leu Ala Phe Glu Asp Ile Tyr Ile Asp Gln Arg Lys Thr Ile  
 1235 1240 1245  
 Lys Thr Met Leu Glu Tyr Ala Asp Lys Val Phe Thr Tyr Ile Phe  
 1250 1255 1260  
 Ile Leu Glu Met Leu Leu Lys Trp Val Ala Tyr Gly Tyr Gln Thr  
 1265 1270 1275  
 Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp Phe Leu Ile Val Asp  
 1280 1285 1290  
 Val Ser Leu Val Ser Leu Thr Ala Asn Ala Leu Gly Tyr Ser Glu  
 1295 1300 1305  
 Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu Arg Ala Leu Arg Pro  
 1310 1315 1320  
 Leu Arg Ala Leu Ser Arg Phe Glu Gly Met Arg Val Val Val Asn  
 1325 1330 1335  
 Ala Leu Leu Gly Ala Ile Pro Ser Ile Met Asn Val Leu Leu Val  
 1340 1345 1350  
 Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile Met Gly Val Asn Leu  
 1355 1360 1365  
 Phe Ala Gly Lys Phe Tyr His Cys Ile Asn Thr Thr Thr Gly Asp  
 1370 1375 1380

## SSCP Update Sequences.ST25

Arg	Phe	Asp	Ile	Glu	Asp	Val	Asn	Asn	His	Thr	Asp	Cys	Leu	Lys
	1385					1390					1395			
Leu	Ile	Glu	Arg	Asn	Glu	Thr	Ala	Arg	Trp	Lys	Asn	Val	Lys	Val
	1400					1405					1410			
Asn	Phe	Asp	Asn	Val	Gly	Phe	Gly	Tyr	Leu	Ser	Leu	Leu	Gln	Val
	1415					1420					1425			
Ala	Thr	Phe	Lys	Gly	Trp	Met	Asp	Ile	Met	Tyr	Ala	Ala	Val	Asp
	1430					1435					1440			
Ser	Arg	Asn	Val	Glu	Leu	Gln	Pro	Lys	Tyr	Glu	Lys	Ser	Leu	Tyr
	1445					1450					1455			
Met	Tyr	Leu	Tyr	Phe	Val	Ile	Phe	Ile	Ile	Phe	Gly	Ser	Phe	Phe
	1460					1465					1470			
Thr	Leu	Asn	Leu	Phe	Ile	Gly	Val	Ile	Ile	Asp	Asn	Phe	Asn	Gln
	1475					1480					1485			
Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile	Phe	Met	Thr	Glu	Glu
	1490					1495					1500			
Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys	Leu	Gly	Ser	Lys	Lys
	1505					1510					1515			
Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Gly	Asn	Lys	Phe	Gln	Gly	Met
	1520					1525					1530			
Val	Phe	Asp	Phe	Val	Thr	Arg	Gln	Val	Phe	Asp	Ile	Ser	Ile	Met
	1535					1540					1545			
Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met	Met	Val	Glu	Thr	Asp
	1550					1555					1560			
Asp	Gln	Ser	Glu	Tyr	Val	Thr	Thr	Ile	Leu	Ser	Arg	Ile	Asn	Leu
	1565					1570					1575			
Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys	Val	Leu	Lys	Leu	Ile
	1580					1585					1590			
Ser	Leu	Arg	His	Tyr	Tyr	Phe	Thr	Ile	Gly	Trp	Asn	Ile	Phe	Asp
	1595					1600					1605			
Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly	Met	Phe	Leu	Ala	Glu
	1610					1615					1620			
Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr	Leu	Phe	Arg	Val	Ile
	1625					1630					1635			



## SSCP Update Sequences.ST25

Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg	Leu	Ile	Lys	Gly	Ala
	1640					1645					1650			
Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu	Met	Met	Ser	Leu	Pro
	1655					1660					1665			
Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe	Leu	Val	Met	Phe	Ile
	1670					1675					1680			
Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala	Tyr	Val	Lys	Arg	Glu
	1685					1690					1695			
Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu	Thr	Phe	Gly	Asn	Ser
	1700					1705					1710			
Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser	Ala	Gly	Trp	Asp	Gly
	1715					1720					1725			
Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Lys	Pro	Pro	Asp	Cys	Asp	Pro
	1730					1735					1740			
Asn	Lys	Val	Asn	Pro	Gly	Ser	Ser	Val	Lys	Gly	Asp	Cys	Gly	Asn
	1745					1750					1755			
Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser	Tyr	Ile	Ile	Ile	Ser
	1760					1765					1770			
Phe	Leu	Val	Val	Val	Asn	Thr	Tyr	Ile	Ala	Val	Ile	Leu	Glu	Asn
	1775					1780					1785			
Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu	Pro	Leu	Ser	Glu	Asp
	1790					1795					1800			
Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu	Lys	Phe	Asp	Pro	Asp
	1805					1810					1815			
Ala	Thr	Gln	Phe	Met	Glu	Phe	Glu	Lys	Leu	Ser	Gln	Phe	Ala	Ala
	1820					1825					1830			
Ala	Leu	Glu	Pro	Pro	Leu	Asn	Leu	Pro	Gln	Pro	Asn	Lys	Leu	Gln
	1835					1840					1845			
Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser	Gly	Asp	Arg	Ile	His
	1850					1855					1860			
Cys	Leu	Asp	Ile	Leu	Phe	Ala	Phe	Thr	Lys	Arg	Val	Leu	Gly	Glu
	1865					1870					1875			
Ser	Gly	Glu	Met	Asp	Ala	Leu	Arg	Ile	Gln	Met	Glu	Glu	Arg	Phe
	1880					1885					1890			

## SSCP Update Sequences.ST25

Met Ala Ser Asn Pro Ser Lys Val Ser Tyr Gln Pro Ile Thr Thr  
1895 1900 1905

Thr Leu Lys Arg Lys Gln Glu Glu Val Ser Ala Val Ile Ile Gln  
1910 1915 1920

Arg Ala Tyr Arg Arg His Leu Leu Lys Arg Thr Val Lys Gln Ala  
1925 1930 1935

Ser Phe Thr Tyr Asn Lys Asn Lys Ile Lys Gly Gly Ala Asn Leu  
1940 1945 1950

Leu Ile Lys Glu Asp Met Ile Ile Asp Arg Ile Asn Glu Asn Ser  
1955 1960 1965

Ile Thr Glu Lys Thr Asp Leu Thr Met Ser Thr Ala Ala Cys Pro  
1970 1975 1980

Pro Ser Tyr Asp Arg Val Thr Lys Pro Ile Val Glu Lys His Glu  
1985 1990 1995

Gln Glu Gly Lys Asp Glu Lys Ala Lys Gly Lys  
2000 2005

<210> 81  
<211> 1891  
<212> PRT  
<213> Homo sapiens

<400> 81

Met Glu Gln Thr Val Leu Val Pro Pro Gly Pro Asp Ser Phe Asn Phe  
1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Arg Arg Ile Ala Glu Glu  
20 25 30

Lys Ala Lys Asn Pro Lys Pro Asp Lys Lys Asp Asp Asp Glu Asn Gly  
35 40 45

Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Asn Leu Pro Phe Ile  
50 55 60

Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Glu Pro Leu Glu Asp Leu  
65 70 75 80

Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys Leu  
85 90 95

Lys Ala Ile Phe Arg Phe Ser Ala Thr Ser Ala Leu Tyr Ile Leu Thr  
100 105 110

## SSCP Update Sequences.ST25

Pro Phe Asn Pro Leu Arg Lys Ile Ala Ile Lys Ile Leu Val His Ser  
 115 120 125  
 Leu Phe Ser Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val Phe  
 130 135 140  
 Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr Thr  
 145 150 155 160  
 Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Ile Ala Arg  
 165 170 175  
 Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn Trp  
 180 185 190  
 Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val Asp  
 195 200 205  
 Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala Leu  
 210 215 220  
 Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala Leu  
 225 230 235 240  
 Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val Phe  
 245 250 255  
 Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly Asn  
 260 265 270  
 Leu Arg Asn Lys Cys Ile Gln Trp Pro Pro Thr Asn Ala Ser Leu Glu  
 275 280 285  
 Glu His Ser Ile Glu Lys Asn Ile Thr Val Asn Tyr Asn Gly Thr Leu  
 290 295 300  
 Ile Asn Glu Thr Val Phe Glu Phe Asp Trp Lys Ser Tyr Ile Gln Asp  
 305 310 315 320  
 Ser Arg Tyr His Tyr Phe Leu Glu Gly Phe Leu Asp Ala Leu Leu Cys  
 325 330 335  
 Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Met Cys Val  
 340 345 350  
 Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp Thr Phe  
 355 360 365  
 Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp Phe Trp  
 370 375 380

## SSCP Update Sequences.ST25

Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr Tyr Met  
 385 390 395 400  
 Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu Ile Asn  
 405 410 415  
 Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn Gln Ala  
 420 425 430  
 Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln Met Ile  
 435 440 445  
 Glu Gln Leu Lys Lys Gln Gln Glu Ala Ala Gln Gln Ala Ala Thr Ala  
 450 455 460  
 Thr Ala Ser Glu His Ser Arg Glu Pro Ser Ala Ala Gly Arg Leu Ser  
 465 470 475 480  
 Asp Ser Ser Ser Glu Ala Ser Lys Leu Ser Ser Lys Ser Ala Lys Glu  
 485 490 495  
 Arg Arg Asn Arg Arg Lys Lys Arg Lys Gln Lys Glu Gln Ser Gly Gly  
 500 505 510  
 Glu Glu Lys Asp Glu Asp Glu Phe Gln Lys Ser Glu Ser Glu Asp Ser  
 515 520 525  
 Ile Arg Arg Lys Gly Phe Arg Phe Ser Ile Glu Gly Asn Arg Leu Thr  
 530 535 540  
 Tyr Glu Lys Arg Tyr Ser Ser Pro His Gln Ser Leu Leu Ser Ile Arg  
 545 550 555 560  
 Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Thr Ser Leu Phe Ser  
 565 570 575  
 Phe Arg Gly Arg Ala Lys Asp Val Gly Ser Glu Asn Asp Phe Ala Asp  
 580 585 590  
 Asp Glu His Ser Thr Phe Glu Asp Asn Glu Ser Arg Arg Asp Ser Leu  
 595 600 605  
 Phe Val Pro Arg Arg His Gly Glu Arg Arg Asn Ser Asn Leu Ser Gln  
 610 615 620  
 Thr Ser Arg Ser Ser Arg Met Leu Ala Val Phe Pro Ala Asn Gly Lys  
 625 630 635 640  
 Met His Ser Thr Val Asp Cys Asn Gly Val Val Ser Leu Val Gly Gly  
 645 650 655

## SSCP Update Sequences.ST25

Pro Ser Val Pro Thr Ser Pro Val Gly Gln Leu Leu Pro Glu Val Ile  
 660 665 670  
 Ile Asp Lys Pro Ala Thr Asp Asp Asn Gly Thr Thr Thr Glu Thr Glu  
 675 680 685  
 Met Arg Lys Arg Arg Ser Ser Ser Phe His Val Ser Met Asp Phe Leu  
 690 695 700  
 Glu Asp Pro Ser Gln Arg Gln Arg Ala Met Ser Ile Ala Ser Ile Leu  
 705 710 715 720  
 Thr Asn Thr Val Glu Glu Leu Glu Glu Ser Arg Gln Lys Cys Pro Pro  
 725 730 735  
 Cys Trp Tyr Lys Phe Ser Asn Ile Phe Leu Ile Trp Asp Cys Ser Pro  
 740 745 750  
 Tyr Trp Leu Lys Val Lys His Val Val Asn Leu Val Val Met Asp Pro  
 755 760 765  
 Phe Val Asp Leu Ala Ile Thr Ile Cys Ile Val Leu Asn Thr Leu Phe  
 770 775 780  
 Met Ala Met Glu His Tyr Pro Met Thr Asp His Phe Asn Asn Val Leu  
 785 790 795 800  
 Thr Val Gly Asn Leu Val Phe Thr Gly Ile Phe Thr Ala Glu Met Phe  
 805 810 815  
 Leu Lys Ile Ile Ala Met Asp Pro Tyr Tyr Tyr Phe Gln Glu Gly Trp  
 820 825 830  
 Asn Ile Phe Asp Gly Phe Ile Val Thr Leu Ser Leu Val Glu Leu Gly  
 835 840 845  
 Leu Ala Asn Val Glu Gly Leu Ser Val Leu Arg Ser Phe Arg Leu Leu  
 850 855 860  
 Arg Val Phe Lys Leu Ala Lys Ser Trp Pro Thr Leu Asn Met Leu Ile  
 865 870 875 880  
 Lys Ile Ile Gly Asn Ser Val Gly Ala Leu Gly Asn Leu Thr Leu Val  
 885 890 895  
 Leu Ala Ile Ile Val Phe Ile Phe Ala Val Val Gly Met Gln Leu Phe  
 900 905 910  
 Gly Lys Ser Tyr Lys Asp Cys Val Cys Lys Ile Ala Ser Asp Cys Gln  
 915 920 925

## SSCP Update Sequences.ST25

Leu Pro Arg Trp His Met Asn Asp Phe Phe His Ser Phe Leu Ile Val  
 930 935 940  
 Phe Arg Val Leu Cys Gly Glu Trp Ile Glu Thr Met Trp Asp Cys Met  
 945 950 955 960  
 Glu Val Ala Gly Gln Ala Met Cys Leu Thr Val Phe Met Met Val Met  
 965 970 975  
 Val Ile Gly Asn Leu Val Val Leu Asn Leu Phe Leu Ala Leu Leu Leu  
 980 985 990  
 Ser Ser Phe Ser Ala Asp Asn Leu Ala Ala Thr Asp Asp Asp Asn Glu  
 995 1000 1005  
 Met Asn Asn Leu Gln Ile Ala Val Asp Arg Met His Lys Gly Val  
 1010 1015 1020  
 Ala Tyr Val Lys Arg Lys Ile Tyr Glu Phe Ile Gln Gln Ser Phe  
 1025 1030 1035  
 Ile Arg Lys Gln Lys Ile Leu Asp Glu Ile Lys Pro Leu Asp Asp  
 1040 1045 1050  
 Leu Asn Asn Lys Lys Asp Ser Cys Met Ser Asn His Thr Thr Glu  
 1055 1060 1065  
 Ile Gly Lys Asp Leu Asp Tyr Leu Lys Asp Val Asn Gly Thr Thr  
 1070 1075 1080  
 Ser Gly Ile Gly Thr Gly Ser Ser Val Glu Lys Tyr Ile Ile Asp  
 1085 1090 1095  
 Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn Pro Ser Leu Thr Val  
 1100 1105 1110  
 Thr Val Pro Ile Ala Val Gly Glu Ser Asp Phe Glu Asn Leu Asn  
 1115 1120 1125  
 Thr Glu Asp Phe Ser Ser Glu Ser Asp Leu Glu Glu Ser Lys Glu  
 1130 1135 1140  
 Lys Leu Asn Glu Ser Ser Ser Ser Ser Glu Gly Ser Thr Val Asp  
 1145 1150 1155  
 Ile Gly Ala Pro Val Glu Glu Gln Pro Val Val Glu Pro Glu Glu  
 1160 1165 1170  
 Thr Leu Glu Pro Glu Ala Cys Phe Thr Glu Gly Cys Val Gln Arg  
 1175 1180 1185

## SSCP Update Sequences.ST25

Phe	Lys	Cys	Cys	Gln	Ile	Asn	Val	Glu	Glu	Gly	Arg	Gly	Lys	Gln
	1190					1195					1200			
Trp	Trp	Asn	Leu	Arg	Arg	Thr	Cys	Phe	Arg	Ile	Val	Glu	His	Asn
	1205					1210					1215			
Trp	Phe	Glu	Thr	Phe	Ile	Val	Phe	Met	Ile	Leu	Leu	Ser	Ser	Gly
	1220					1225					1230			
Ala	Leu	Ala	Phe	Glu	Asp	Ile	Tyr	Ile	Asp	Gln	Arg	Lys	Thr	Ile
	1235					1240					1245			
Lys	Thr	Met	Leu	Glu	Tyr	Ala	Asp	Lys	Val	Phe	Thr	Tyr	Ile	Phe
	1250					1255					1260			
Ile	Leu	Glu	Met	Leu	Leu	Lys	Trp	Val	Ala	Tyr	Gly	Tyr	Gln	Thr
	1265					1270					1275			
Tyr	Phe	Thr	Asn	Ala	Trp	Cys	Trp	Leu	Asp	Phe	Leu	Ile	Val	Asp
	1280					1285					1290			
Val	Ser	Leu	Val	Ser	Leu	Thr	Ala	Asn	Ala	Leu	Gly	Tyr	Ser	Glu
	1295					1300					1305			
Leu	Gly	Ala	Ile	Lys	Ser	Leu	Arg	Thr	Leu	Arg	Ala	Leu	Arg	Pro
	1310					1315					1320			
Leu	Arg	Ala	Leu	Ser	Arg	Phe	Glu	Gly	Met	Arg	Val	Val	Val	Asn
	1325					1330					1335			
Ala	Leu	Leu	Gly	Ala	Ile	Pro	Ser	Ile	Met	Asn	Val	Leu	Leu	Val
	1340					1345					1350			
Cys	Leu	Ile	Phe	Trp	Leu	Ile	Phe	Ser	Ile	Met	Gly	Val	Asn	Leu
	1355					1360					1365			
Phe	Ala	Gly	Lys	Phe	Tyr	His	Cys	Ile	Asn	Thr	Thr	Thr	Gly	Asp
	1370					1375					1380			
Arg	Phe	Asp	Ile	Glu	Asp	Val	Asn	Asn	His	Thr	Asp	Cys	Leu	Lys
	1385					1390					1395			
Leu	Ile	Glu	Arg	Asn	Glu	Thr	Ala	Arg	Trp	Lys	Asn	Val	Lys	Val
	1400					1405					1410			
Asn	Phe	Asp	Asn	Val	Gly	Phe	Gly	Tyr	Leu	Ser	Leu	Leu	Gln	Val
	1415					1420					1425			
Ala	Thr	Phe	Lys	Gly	Trp	Met	Asp	Ile	Met	Tyr	Ala	Ala	Val	Asp
	1430					1435					1440			

## SSCP Update Sequences.ST25

Ser	Arg	Asn	Val	Glu	Leu	Gln	Pro	Lys	Tyr	Glu	Lys	Ser	Leu	Tyr
	1445					1450					1455			
Met	Tyr	Leu	Tyr	Phe	Val	Ile	Phe	Ile	Ile	Phe	Gly	Ser	Phe	Phe
	1460					1465					1470			
Thr	Leu	Asn	Leu	Phe	Ile	Gly	Val	Ile	Ile	Asp	Asn	Phe	Asn	Gln
	1475					1480					1485			
Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile	Phe	Met	Thr	Glu	Glu
	1490					1495					1500			
Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys	Leu	Gly	Ser	Lys	Lys
	1505					1510					1515			
Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Gly	Asn	Lys	Phe	Gln	Gly	Met
	1520					1525					1530			
Val	Phe	Asp	Phe	Val	Thr	Arg	Gln	Val	Phe	Asp	Ile	Ser	Ile	Met
	1535					1540					1545			
Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met	Met	Val	Glu	Thr	Asp
	1550					1555					1560			
Asp	Gln	Ser	Glu	Tyr	Val	Thr	Thr	Ile	Leu	Ser	Arg	Ile	Asn	Leu
	1565					1570					1575			
Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys	Val	Leu	Lys	Leu	Ile
	1580					1585					1590			
Ser	Leu	Arg	His	Tyr	Tyr	Phe	Thr	Ile	Gly	Trp	Asn	Ile	Phe	Asp
	1595					1600					1605			
Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly	Met	Phe	Leu	Ala	Glu
	1610					1615					1620			
Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr	Leu	Phe	Arg	Val	Ile
	1625					1630					1635			
Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg	Leu	Ile	Lys	Gly	Ala
	1640					1645					1650			
Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu	Met	Met	Ser	Leu	Pro
	1655					1660					1665			
Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe	Leu	Val	Met	Phe	Ile
	1670					1675					1680			
Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala	Tyr	Val	Lys	Arg	Glu
	1685					1690					1695			



## SSCP Update Sequences.ST25

Val Gly Ile Asp Asp Met Phe Asn Phe Glu Thr Phe Gly Asn Ser  
 1700 1705 1710  
 Met Ile Cys Leu Phe Gln Ile Thr Thr Ser Ala Gly Trp Asp Gly  
 1715 1720 1725  
 Leu Leu Ala Pro Ile Leu Asn Ser Lys Pro Pro Asp Cys Asp Pro  
 1730 1735 1740  
 Asn Lys Val Asn Pro Gly Ser Ser Val Lys Gly Asp Cys Gly Asn  
 1745 1750 1755  
 Pro Ser Val Gly Ile Phe Phe Phe Val Ser Tyr Ile Ile Ile Ser  
 1760 1765 1770  
 Phe Leu Val Val Val Asn Met Tyr Ile Ala Val Ile Leu Glu Asn  
 1775 1780 1785  
 Phe Ser Val Ala Thr Glu Glu Ser Ala Glu Pro Leu Ser Glu Asp  
 1790 1795 1800  
 Asp Phe Glu Met Phe Tyr Glu Val Trp Glu Lys Phe Asp Pro Asp  
 1805 1810 1815  
 Ala Thr Gln Phe Met Glu Phe Glu Lys Leu Ser Gln Phe Ala Ala  
 1820 1825 1830  
 Ala Leu Glu Pro Pro Leu Asn Leu Pro Gln Pro Asn Lys Leu Gln  
 1835 1840 1845  
 Leu Ile Ala Met Asp Leu Pro Met Val Ser Gly Asp Arg Ile His  
 1850 1855 1860  
 Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys Arg Val Leu Gly Glu  
 1865 1870 1875  
 Ser Gly Glu Met Asp Ala Leu Arg Ile Gln Met Glu Glu  
 1880 1885 1890  
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 <211> 218  
 <212> PRT  
 <213> Homo sapiens  
 <400> 82  
 Met Gly Arg Leu Leu Ala Leu Val Val Gly Ala Ala Leu Val Ser Ser  
 1 5 10 15  
 Ala Cys Gly Gly Cys Val Glu Val Asp Ser Glu Thr Glu Ala Val Tyr  
 20 25 30

## SSCP Update Sequences.ST25

Gly Met Thr Phe Lys Ile Leu Cys Ile Ser Cys Lys Arg Arg Ser Glu  
 35 40 45

Thr Asn Ala Glu Thr Phe Thr Glu Trp Thr Phe Arg Gln Lys Gly Thr  
 50 55 60

Glu Glu Phe Val Lys Ile Leu Arg Tyr Glu Asn Glu Val Leu Gln Leu  
 65 70 75 80

Glu Glu Asp Glu His Phe Glu Gly Arg Val Val Trp Asn Gly Ser Arg  
 85 90 95

Gly Thr Lys Asp Leu Gln Asp Leu Ser Ile Phe Ile Thr Asn Val Thr  
 100 105 110

Tyr Asn His Ser Gly Asp Tyr Glu Cys His Val Tyr Arg Leu Leu Phe  
 115 120 125

Phe Glu Asn Tyr Glu His Asn Thr Ser Val Val Lys Lys Ile His Ile  
 130 135 140

Glu Val Val Asp Lys Ala Asn Arg Asp Met Ala Ser Ile Val Ser Glu  
 145 150 155 160

Ile Met Met Tyr Val Leu Ile Val Val Leu Thr Ile Trp Leu Val Ala  
 165 170 175

Glu Met Ile Tyr Cys Tyr Lys Lys Ile Ala Ala Ala Thr Glu Thr Ala  
 180 185 190

Ala Gln Glu Asn Ala Ser Glu Tyr Leu Ala Ile Thr Ser Glu Ser Lys  
 195 200 205

Glu Asn Cys Thr Gly Val Gln Val Ala Glu  
 210 215

<210> 83  
 <211> 2005  
 <212> PRT  
 <213> Homo sapiens

<400> 83

Met Ala Gln Ser Val Leu Val Pro Pro Gly Pro Asp Ser Phe Arg Phe  
 1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Gln Arg Ile Ala Glu Glu  
 20 25 30

Lys Ala Lys Arg Pro Lys Gln Glu Arg Lys Asp Glu Asp Asp Glu Asn  
 35 40 45

Gly Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Ser Leu Pro Phe  
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## SSCP Update Sequences.ST25

50

55

60

Ile Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Val Pro Leu Glu Asp  
65 70 75 80

Leu Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys  
85 90 95

Gly Lys Ala Ile Ser Arg Phe Ser Ala Thr Pro Ala Leu Tyr Ile Leu  
100 105 110

Thr Pro Phe Asn Pro Ile Arg Lys Leu Ala Ile Lys Ile Leu Val His  
115 120 125

Ser Leu Phe Asn Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val  
130 135 140

Phe Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr  
145 150 155 160

Thr Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Leu Ala  
165 170 175

Arg Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn  
180 185 190

Trp Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val  
195 200 205

Asp Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Gln Ala  
210 215 220

Leu Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala  
225 230 235 240

Leu Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val  
245 250 255

Phe Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly  
260 265 270

Asn Leu Arg Asn Lys Cys Leu Gln Trp Pro Pro Asp Asn Ser Ser Phe  
275 280 285

Glu Ile Asn Ile Thr Ser Phe Phe Asn Asn Ser Leu Asp Gly Asn Gly  
290 295 300

Thr Thr Phe Asn Arg Thr Val Ser Ile Phe Asn Trp Asp Glu Tyr Ile  
305 310 315 320

Glu Asp Lys Ser His Phe Tyr Phe Leu Glu Gly Gln Asn Asp Ala Leu  
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## SSCP Update Sequences.ST25

325

330

335

Leu Cys Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Ile  
 340 345 350

Cys Val Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp  
 355 360 365

Thr Phe Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp  
 370 375 380

Phe Trp Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr  
 385 390 395 400

Tyr Met Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu  
 405 410 415

Ile Asn Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn  
 420 425 430

Gln Ala Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln  
 435 440 445

Met Leu Glu Gln Leu Lys Lys Gln Gln Glu Glu Ala Gln Ala Ala Ala  
 450 455 460

Ala Ala Ala Ser Ala Glu Ser Arg Asp Phe Ser Gly Ala Gly Gly Ile  
 465 470 475 480

Gly Val Phe Ser Glu Ser Ser Ser Val Ala Ser Lys Leu Ser Ser Lys  
 485 490 495

Ser Glu Lys Glu Leu Lys Asn Arg Arg Lys Lys Lys Lys Gln Lys Glu  
 500 505 510

Gln Ser Gly Glu Glu Glu Lys Asn Asp Arg Val Leu Lys Ser Glu Ser  
 515 520 525

Glu Asp Ser Ile Arg Arg Lys Gly Phe Arg Phe Ser Leu Glu Gly Ser  
 530 535 540

Arg Leu Thr Tyr Glu Lys Arg Phe Ser Ser Pro His Gln Ser Leu Leu  
 545 550 555 560

Ser Ile Arg Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Ala Ser  
 565 570 575

Leu Phe Ser Phe Arg Gly Arg Ala Lys Asp Ile Gly Ser Glu Asn Asp  
 580 585 590

Phe Ala Asp Asp Glu His Ser Thr Phe Glu Asp Asn Asp Ser Arg Arg

SSCP Update Sequences.ST25  
600 605

595

Asp Ser Leu Phe Val Pro His Arg His Gly Glu Arg Arg His Ser Asn  
 610 615 620  
 Val Ser Gln Ala Ser Arg Ala Ser Arg Val Leu Pro Ile Leu Pro Met  
 625 630 635 640  
 Asn Gly Lys Met His Ser Ala Val Asp Cys Asn Gly Val Val Ser Leu  
 645 650 655  
 Val Gly Gly Pro Ser Thr Leu Thr Ser Ala Gly Gln Leu Leu Pro Glu  
 660 665 670  
 Gly Thr Thr Thr Glu Thr Glu Ile Arg Lys Arg Arg Ser Ser Ser Tyr  
 675 680 685  
 His Val Ser Met Asp Leu Leu Glu Asp Pro Thr Ser Arg Gln Arg Ala  
 690 695 700  
 Met Ser Ile Ala Ser Ile Leu Thr Asn Thr Met Glu Glu Leu Glu Glu  
 705 710 715 720  
 Ser Arg Gln Lys Cys Pro Pro Cys Trp Tyr Lys Phe Ala Asn Met Cys  
 725 730 735  
 Leu Ile Trp Asp Cys Cys Lys Pro Trp Leu Lys Val Lys His Leu Val  
 740 745 750  
 Asn Leu Val Val Met Asp Pro Phe Val Asp Leu Ala Ile Thr Ile Cys  
 755 760 765  
 Ile Val Leu Asn Thr Leu Phe Met Ala Met Glu His Tyr Pro Met Thr  
 770 775 780  
 Glu Gln Phe Ser Ser Val Leu Ser Val Gly Asn Leu Val Phe Thr Gly  
 785 790 795 800  
 Ile Phe Thr Ala Glu Met Phe Leu Lys Ile Ile Ala Met Asp Pro Tyr  
 805 810 815  
 Tyr Tyr Phe Gln Glu Gly Trp Asn Ile Phe Asp Gly Phe Ile Val Ser  
 820 825 830  
 Leu Ser Leu Met Glu Leu Gly Leu Ala Asn Val Glu Gly Leu Ser Val  
 835 840 845  
 Leu Arg Ser Phe Arg Leu Leu Arg Val Phe Lys Leu Ala Lys Ser Trp  
 850 855 860  
 Pro Thr Leu Asn Met Leu Ile Lys Ile Ile Gly Asn Ser Val Gly Ala

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## SSCP Update Sequences ST25

1130						1135						1140		
Ser	Thr	Val	Asp	Ile	Gly	Ala	Pro	Ala	Glu	Gly	Glu	Gln	Pro	Glu
1145						1150					1155			
Val	Glu	Pro	Glu	Glu	Ser	Leu	Glu	Pro	Glu	Ala	Cys	Phe	Thr	Glu
1160						1165					1170			
Asp	Cys	Val	Arg	Lys	Phe	Lys	Cys	Cys	Gln	Ile	Ser	Ile	Glu	Glu
1175						1180					1185			
Gly	Lys	Gly	Lys	Leu	Trp	Trp	Asn	Leu	Arg	Lys	Thr	Cys	Tyr	Lys
1190						1195					1200			
Ile	Val	Glu	His	Asn	Trp	Phe	Glu	Thr	Phe	Ile	Val	Phe	Met	Ile
1205						1210					1215			
Leu	Leu	Ser	Ser	Gly	Ala	Leu	Ala	Phe	Glu	Asp	Ile	Tyr	Ile	Glu
1220						1225					1230			
Gln	Arg	Lys	Thr	Ile	Lys	Thr	Met	Leu	Glu	Tyr	Ala	Asp	Lys	Val
1235						1240					1245			
Phe	Thr	Tyr	Ile	Phe	Ile	Leu	Glu	Met	Leu	Leu	Lys	Trp	Val	Ala
1250						1255					1260			
Tyr	Gly	Phe	Gln	Val	Tyr	Phe	Thr	Asn	Ala	Trp	Cys	Trp	Leu	Asp
1265						1270					1275			
Phe	Leu	Ile	Val	Asp	Val	Ser	Leu	Val	Ser	Leu	Thr	Ala	Asn	Ala
1280						1285					1290			
Leu	Gly	Tyr	Ser	Glu	Leu	Gly	Ala	Ile	Lys	Ser	Leu	Arg	Thr	Leu
1295						1300					1305			
Arg	Ala	Leu	Arg	Pro	Leu	Arg	Ala	Leu	Ser	Arg	Phe	Glu	Gly	Met
1310						1315					1320			
Arg	Val	Val	Val	Asn	Ala	Leu	Leu	Gly	Ala	Ile	Pro	Ser	Ile	Met
1325						1330					1335			
Asn	Val	Leu	Leu	Val	Cys	Leu	Ile	Phe	Trp	Leu	Ile	Phe	Ser	Ile
1340						1345					1350			
Met	Gly	Val	Asn	Leu	Phe	Ala	Gly	Lys	Phe	Tyr	His	Cys	Ile	Asn
1355						1360					1365			
Tyr	Thr	Thr	Gly	Glu	Met	Phe	Asp	Val	Ser	Val	Val	Asn	Asn	Tyr
1370						1375					1380			
Ser	Glu	Cys	Lys	Ala	Leu	Ile	Glu	Ser	Asn	Gln	Thr	Ala	Arg	Trp

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## SSCP Update Sequences ST25

1640														
Met	Met	Ser	Leu	Pro	Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe
	1655					1660					1665			
Leu	Val	Met	Phe	Ile	Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala
	1670					1675					1680			
Tyr	Val	Lys	Arg	Glu	Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu
	1685					1690					1695			
Thr	Phe	Gly	Asn	Ser	Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser
	1700					1705					1710			
Ala	Gly	Trp	Asp	Gly	Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Gly	Pro
	1715					1720					1725			
Pro	Asp	Cys	Asp	Pro	Asp	Lys	Asp	His	Pro	Gly	Ser	Ser	Val	Lys
	1730					1735					1740			
Gly	Asp	Cys	Gly	Asn	Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser
	1745					1750					1755			
Tyr	Ile	Ile	Ile	Ser	Phe	Leu	Val	Val	Leu	Asn	Met	Tyr	Ile	Ala
	1760					1765					1770			
Val	Ile	Leu	Glu	Asn	Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu
	1775					1780					1785			
Pro	Leu	Ser	Glu	Asp	Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu
	1790					1795					1800			
Lys	Phe	Asp	Pro	Asp	Ala	Thr	Gln	Phe	Ile	Glu	Phe	Ala	Lys	Leu
	1805					1810					1815			
Ser	Asp	Phe	Ala	Asp	Ala	Leu	Asp	Pro	Pro	Leu	Leu	Ile	Ala	Lys
	1820					1825					1830			
Pro	Asn	Lys	Val	Gln	Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser
	1835					1840					1845			
Gly	Asp	Arg	Ile	His	Cys	Leu	Asp	Ile	Leu	Phe	Ala	Phe	Thr	Lys
	1850					1855					1860			
Arg	Val	Leu	Gly	Glu	Ser	Gly	Glu	Met	Asp	Ala	Leu	Arg	Ile	Gln
	1865					1870					1875			
Met	Glu	Glu	Arg	Phe	Met	Ala	Ser	Asn	Pro	Ser	Lys	Val	Ser	Tyr
	1880					1885					1890			
Glu	Pro	Ile	Thr	Thr	Thr	Leu	Lys	Arg	Lys	Gln	Glu	Glu	Val	Ser

## SSCP Update Sequences.ST25

1895 1900 1905

Ala Ile Ile Ile Gln Arg Ala Tyr Arg Arg Tyr Leu Leu Lys Gln  
1910 1915 1920

Lys Val Lys Lys Val Ser Ser Ile Tyr Lys Lys Asp Lys Gly Lys  
1925 1930 1935

Glu Cys Asp Gly Thr Pro Ile Lys Glu Asp Thr Leu Ile Asp Lys  
1940 1945 1950

Leu Asn Glu Asn Ser Thr Pro Glu Lys Thr Asp Met Thr Pro Ser  
1955 1960 1965

Thr Thr Ser Pro Pro Ser Tyr Asp Ser Val Thr Lys Pro Glu Lys  
1970 1975 1980

Glu Lys Phe Glu Lys Asp Lys Ser Glu Lys Glu Asp Lys Gly Lys  
1985 1990 1995

Asp Ile Arg Glu Ser Lys Lys  
2000 2005

<210> 84  
<211> 2005  
<212> PRT  
<213> Homo sapiens

<400> 84

Met Ala Gln Ser Val Leu Val Pro Pro Gly Pro Asp Ser Phe Arg Phe  
1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Gln Arg Ile Ala Glu Glu  
20 25 30

Lys Ala Lys Arg Pro Lys Gln Glu Arg Lys Asp Glu Asp Asp Glu Asn  
35 40 45

Gly Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Ser Leu Pro Phe  
50 55 60

Ile Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Val Pro Leu Glu Asp  
65 70 75 80

Leu Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys  
85 90 95

Gly Lys Ala Ile Ser Arg Phe Ser Ala Thr Pro Ala Leu Tyr Ile Leu  
100 105 110

Thr Pro Phe Asn Pro Ile Arg Lys Leu Ala Ile Lys Ile Leu Val His  
115 120 125

## SSCP Update Sequences.ST25

Ser Leu Phe Asn Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val  
 130 135 140  
 Phe Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr  
 145 150 155 160  
 Thr Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Leu Ala  
 165 170 175  
 Arg Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn  
 180 185 190  
 Trp Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val  
 195 200 205  
 Asp Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala  
 210 215 220  
 Leu Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala  
 225 230 235 240  
 Leu Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val  
 245 250 255  
 Phe Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly  
 260 265 270  
 Asn Leu Arg Asn Lys Cys Leu Gln Trp Pro Pro Asp Asn Ser Ser Phe  
 275 280 285  
 Glu Ile Asn Ile Thr Ser Phe Phe Asn Asn Ser Leu Asp Gly Asn Gly  
 290 295 300  
 Thr Thr Phe Asn Arg Thr Val Ser Ile Phe Asn Trp Asp Glu Tyr Ile  
 305 310 315 320  
 Glu Asp Lys Ser His Phe Tyr Phe Leu Glu Gly Gln Asn Asp Ala Leu  
 325 330 335  
 Leu Cys Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Ile  
 340 345 350  
 Cys Val Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp  
 355 360 365  
 Thr Phe Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp  
 370 375 380  
 Phe Trp Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr  
 385 390 395 400

## SSCP Update Sequences.ST25

Tyr Met Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu  
 405 410 415  
 Ile Asn Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn  
 420 425 430  
 Gln Ala Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln  
 435 440 445  
 Met Leu Glu Gln Leu Lys Lys Gln Gln Glu Glu Ala Gln Ala Ala Ala  
 450 455 460  
 Ala Ala Ala Ser Ala Glu Ser Arg Asp Phe Ser Gly Ala Gly Gly Ile  
 465 470 475 480  
 Gly Val Phe Ser Glu Ser Ser Ser Val Ala Ser Lys Leu Ser Ser Lys  
 485 490 495  
 Ser Glu Lys Glu Leu Lys Asn Arg Arg Lys Lys Lys Lys Gln Lys Glu  
 500 505 510  
 Gln Ser Gly Glu Glu Glu Lys Asn Asp Arg Val Leu Lys Ser Glu Ser  
 515 520 525  
 Glu Asp Ser Ile Arg Arg Lys Gly Phe Arg Phe Ser Leu Glu Gly Ser  
 530 535 540  
 Arg Leu Thr Tyr Glu Lys Arg Phe Ser Ser Pro His Gln Ser Leu Leu  
 545 550 555 560  
 Ser Ile Arg Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Ala Ser  
 565 570 575  
 Leu Phe Ser Phe Arg Gly Arg Ala Lys Asp Ile Gly Ser Glu Asn Asp  
 580 585 590  
 Phe Ala Asp Asp Glu His Ser Thr Phe Glu Asp Asn Asp Ser Arg Arg  
 595 600 605  
 Asp Ser Leu Phe Val Pro His Arg His Gly Glu Arg Arg His Ser Asn  
 610 615 620  
 Val Ser Gln Ala Ser Arg Ala Ser Arg Val Leu Pro Ile Leu Pro Met  
 625 630 635 640  
 Asn Gly Lys Met His Ser Ala Val Asp Cys Asn Gly Val Val Ser Leu  
 645 650 655  
 Val Gly Gly Pro Ser Thr Leu Thr Ser Ala Gly Gln Leu Leu Pro Glu  
 660 665 670

## SSCP update Sequences.ST25

Gly Thr Thr Thr Glu Thr Glu Ile Arg Lys Arg Arg Ser Ser Ser Tyr  
 675 680 685  
 His Val Ser Met Asp Leu Leu Glu Asp Pro Thr Ser Arg Gln Arg Ala  
 690 695 700  
 Met Ser Ile Ala Ser Ile Leu Thr Asn Thr Met Glu Glu Leu Glu Glu  
 705 710 715 720  
 Ser Arg Gln Lys Cys Pro Pro Cys Trp Tyr Lys Phe Ala Asn Met Cys  
 725 730 735  
 Leu Ile Trp Asp Cys Cys Lys Pro Trp Leu Lys Val Lys His Leu Val  
 740 745 750  
 Asn Leu Val Val Met Asp Pro Phe Val Asp Leu Ala Ile Thr Ile Cys  
 755 760 765  
 Ile Val Leu Asn Thr Leu Phe Met Ala Met Glu His Tyr Pro Met Thr  
 770 775 780  
 Glu Gln Phe Ser Ser Val Leu Ser Val Gly Asn Leu Val Phe Thr Gly  
 785 790 795 800  
 Ile Phe Thr Ala Glu Met Phe Leu Lys Ile Ile Ala Met Asp Pro Tyr  
 805 810 815  
 Tyr Tyr Phe Gln Glu Gly Trp Asn Ile Phe Asp Gly Phe Ile Val Ser  
 820 825 830  
 Leu Ser Leu Met Glu Leu Gly Leu Ala Asn Val Glu Gly Leu Ser Val  
 835 840 845  
 Leu Arg Ser Phe Arg Leu Leu Arg Val Phe Lys Leu Ala Lys Ser Trp  
 850 855 860  
 Pro Thr Leu Asn Met Leu Ile Lys Ile Ile Gly Asn Ser Val Gly Ala  
 865 870 875 880  
 Leu Gly Asn Leu Thr Leu Val Leu Ala Ile Ile Ile Phe Ile Phe Ala  
 885 890 895  
 Val Val Gly Met Gln Leu Phe Gly Lys Ser Tyr Lys Glu Cys Val Cys  
 900 905 910  
 Lys Ile Ser Asn Asp Cys Glu Leu Pro Arg Trp His Met His Asp Phe  
 915 920 925  
 Phe His Ser Phe Leu Ile Val Phe Arg Val Leu Cys Gly Glu Trp Ile  
 930 935 940

## SSCP Update Sequences.ST25

Glu Thr Met Trp Asp Cys Met Glu Val Ala Gly Gln Thr Met Cys Leu  
 945 950 955 960  
 Thr Val Phe Met Met Val Met Val Ile Gly Asn Leu Val Val Leu Asn  
 965 970 975  
 Leu Phe Leu Ala Leu Leu Leu Ser Ser Phe Ser Ser Asp Asn Leu Ala  
 980 985 990  
 Ala Thr Asp Asp Asp Asn Glu Met Asn Asn Leu Gln Ile Ala Val Gly  
 995 1000 1005  
 Arg Met Gln Lys Gly Ile Asp Phe Val Lys Arg Lys Ile Arg Glu  
 1010 1015 1020  
 Phe Ile Gln Lys Ala Phe Val Arg Lys Gln Lys Ala Leu Asp Glu  
 1025 1030 1035  
 Ile Lys Pro Leu Glu Asp Leu Asn Asn Lys Lys Asp Ser Cys Ile  
 1040 1045 1050  
 Ser Asn His Thr Thr Ile Glu Ile Gly Lys Asp Leu Asn Tyr Leu  
 1055 1060 1065  
 Lys Asp Gly Asn Gly Thr Thr Ser Gly Ile Gly Ser Ser Val Glu  
 1070 1075 1080  
 Lys Tyr Val Val Asp Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn  
 1085 1090 1095  
 Pro Ser Leu Thr Val Thr Val Pro Ile Ala Val Gly Glu Ser Asp  
 1100 1105 1110  
 Phe Glu Asn Leu Asn Thr Glu Glu Phe Ser Ser Glu Ser Asp Met  
 1115 1120 1125  
 Glu Glu Ser Lys Glu Lys Leu Asn Ala Thr Ser Ser Ser Glu Gly  
 1130 1135 1140  
 Ser Thr Val Asp Ile Gly Ala Pro Ala Glu Gly Glu Gln Pro Glu  
 1145 1150 1155  
 Val Glu Pro Glu Glu Ser Leu Glu Pro Glu Ala Cys Phe Thr Glu  
 1160 1165 1170  
 Asp Cys Val Arg Lys Phe Lys Cys Cys Gln Ile Ser Ile Glu Glu  
 1175 1180 1185  
 Gly Lys Gly Lys Leu Trp Trp Asn Leu Arg Lys Thr Cys Tyr Lys  
 1190 1195 1200

## SSCP update Sequences.ST25

Ile	Val	Glu	His	Asn	Trp	Phe	Glu	Thr	Phe	Ile	Val	Phe	Met	Ile
	1205					1210					1215			
Leu	Leu	Ser	Ser	Gly	Ala	Leu	Ala	Phe	Glu	Asp	Ile	Tyr	Ile	Glu
	1220					1225					1230			
Gln	Arg	Lys	Thr	Ile	Lys	Thr	Met	Leu	Glu	Tyr	Ala	Asp	Lys	Val
	1235					1240					1245			
Phe	Thr	Tyr	Ile	Phe	Ile	Leu	Glu	Met	Leu	Leu	Lys	Trp	Val	Ala
	1250					1255					1260			
Tyr	Gly	Phe	Gln	Val	Tyr	Phe	Thr	Asn	Ala	Trp	Cys	Trp	Leu	Asp
	1265					1270					1275			
Phe	Leu	Ile	Val	Asp	Val	Ser	Leu	Val	Ser	Leu	Thr	Ala	Asn	Ala
	1280					1285					1290			
Leu	Gly	Tyr	Ser	Glu	Leu	Gly	Ala	Ile	Lys	Ser	Leu	Arg	Thr	Leu
	1295					1300					1305			
Arg	Ala	Leu	Arg	Pro	Leu	Arg	Ala	Leu	Ser	Arg	Phe	Glu	Gly	Met
	1310					1315					1320			
Arg	Val	Val	Val	Asn	Ala	Leu	Leu	Gly	Ala	Ile	Pro	Ser	Ile	Met
	1325					1330					1335			
Asn	Val	Leu	Leu	Val	Cys	Leu	Ile	Phe	Trp	Leu	Ile	Phe	Ser	Ile
	1340					1345					1350			
Met	Gly	Val	Asn	Leu	Phe	Ala	Gly	Lys	Phe	Tyr	His	Cys	Ile	Asn
	1355					1360					1365			
Tyr	Thr	Thr	Gly	Glu	Met	Phe	Asp	Val	Ser	Val	Val	Asn	Asn	Tyr
	1370					1375					1380			
Ser	Glu	Cys	Lys	Ala	Leu	Ile	Glu	Ser	Asn	Gln	Thr	Ala	Arg	Trp
	1385					1390					1395			
Lys	Asn	Val	Lys	Val	Asn	Phe	Asp	Asn	Val	Gly	Leu	Gly	Tyr	Leu
	1400					1405					1410			
Ser	Leu	Leu	Gln	Val	Ala	Thr	Phe	Lys	Gly	Trp	Met	Asp	Ile	Met
	1415					1420					1425			
Tyr	Ala	Ala	Val	Asp	Ser	Arg	Asn	Val	Glu	Leu	Gln	Pro	Lys	Tyr
	1430					1435					1440			
Glu	Asp	Asn	Leu	Tyr	Met	Tyr	Leu	Tyr	Phe	Val	Ile	Phe	Ile	Ile
	1445					1450					1455			

## SSCP Update Sequences.ST25

Phe	Gly	Ser	Phe	Phe	Thr	Leu	Asn	Leu	Phe	Ile	Gly	Val	Ile	Ile
	1460					1465					1470			
Asp	Asn	Phe	Asn	Gln	Gln	Lys	Lys	Lys	Phe	Gly	Gly	Gln	Asp	Ile
	1475					1480					1485			
Phe	Met	Thr	Glu	Glu	Gln	Lys	Lys	Tyr	Tyr	Asn	Ala	Met	Lys	Lys
	1490					1495					1500			
Leu	Gly	Ser	Lys	Lys	Pro	Gln	Lys	Pro	Ile	Pro	Arg	Pro	Ala	Asn
	1505					1510					1515			
Lys	Phe	Gln	Gly	Met	Val	Phe	Asp	Phe	Val	Thr	Lys	Gln	Val	Phe
	1520					1525					1530			
Asp	Ile	Ser	Ile	Met	Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met
	1535					1540					1545			
Met	Val	Glu	Thr	Asp	Asp	Gln	Ser	Gln	Glu	Met	Thr	Asn	Ile	Leu
	1550					1555					1560			
Tyr	Trp	Ile	Asn	Leu	Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys
	1565					1570					1575			
Val	Leu	Lys	Leu	Ile	Ser	Leu	Arg	Tyr	Tyr	Tyr	Phe	Thr	Ile	Gly
	1580					1585					1590			
Trp	Asn	Ile	Phe	Asp	Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly
	1595					1600					1605			
Met	Phe	Leu	Ala	Glu	Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr
	1610					1615					1620			
Leu	Phe	Arg	Val	Ile	Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg
	1625					1630					1635			
Leu	Ile	Lys	Gly	Ala	Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu
	1640					1645					1650			
Met	Met	Ser	Leu	Pro	Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe
	1655					1660					1665			
Leu	Val	Met	Phe	Ile	Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala
	1670					1675					1680			
Tyr	Val	Lys	Arg	Glu	Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu
	1685					1690					1695			
Thr	Phe	Gly	Asn	Ser	Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser
	1700					1705					1710			



## SSCP Update Sequences.ST25

Ala Gly Trp Asp Gly Leu Leu Ala Pro Ile Leu Asn Ser Gly Pro  
 1715 1720 1725  
 ,  
 Pro Asp Cys Asp Pro Asp Lys Asp His Pro Gly Ser Ser Val Lys  
 1730 1735 1740  
 Gly Asp Cys Gly Asn Pro Ser Val Gly Ile Phe Phe Phe Val Ser  
 1745 1750 1755  
 Tyr Ile Ile Ile Ser Phe Leu Val Val Leu Asn Met Tyr Ile Ala  
 1760 1765 1770  
 Val Ile Leu Glu Asn Phe Ser Val Ala Thr Glu Glu Ser Ala Glu  
 1775 1780 1785  
 Pro Leu Ser Glu Asp Asp Phe Glu Met Phe Tyr Glu Val Trp Glu  
 1790 1795 1800  
 Lys Phe Asp Pro Asp Ala Thr Gln Phe Ile Glu Phe Ala Lys Leu  
 1805 1810 1815  
 Ser Asp Phe Ala Asp Ala Leu Asp Pro Pro Leu Leu Ile Ala Lys  
 1820 1825 1830  
 Pro Asn Lys Val Gln Leu Ile Ala Met Asp Leu Pro Met Val Ser  
 1835 1840 1845  
 Gly Asp Arg Ile His Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys  
 1850 1855 1860  
 Arg Val Leu Gly Glu Ser Gly Glu Met Asp Ala Leu Arg Ile Gln  
 1865 1870 1875  
 Met Glu Glu Arg Phe Met Ala Ser Asn Pro Ser Lys Val Ser Tyr  
 1880 1885 1890  
 Glu Pro Ile Thr Thr Thr Leu Lys Arg Lys Gln Glu Glu Val Ser  
 1895 1900 1905  
 Ala Ile Ile Ile Gln Arg Ala Tyr Arg Arg Tyr Leu Leu Lys Gln  
 1910 1915 1920  
 Lys Val Lys Lys Val Ser Ser Ile Tyr Lys Lys Asp Lys Gly Lys  
 1925 1930 1935  
 Glu Cys Asp Gly Thr Pro Ile Lys Glu Asp Thr Leu Ile Asp Lys  
 1940 1945 1950  
 Leu Asn Glu Asn Ser Thr Pro Glu Lys Thr Asp Met Thr Pro Ser  
 1955 1960 1965

## SSCP Update Sequences.ST25

Thr Thr Ser Pro Pro Ser Tyr Asp Ser Val Thr Lys Pro Glu Lys  
 1970 1975 1980

Glu Lys Phe Glu Lys Asp Lys Ser Glu Lys Glu Asp Lys Gly Lys  
 1985 1990 1995

Asp Ile Arg Glu Ser Lys Lys  
 2000 2005

<210> 85  
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 <212> PRT  
 <213> Homo sapiens

<400> 85

Met Ala Gln Ser Val Leu Val Pro Pro Gly Pro Asp Ser Phe Arg Phe  
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Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Gln Arg Ile Ala Glu Glu  
 20 25 30

Lys Ala Lys Arg Pro Lys Gln Glu Arg Lys Asp Glu Asp Asp Glu Asn  
 35 40 45

Gly Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Ser Leu Pro Phe  
 50 55 60

Ile Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Val Pro Leu Glu Asp  
 65 70 75 80

Leu Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys  
 85 90 95

Gly Lys Ala Ile Ser Arg Phe Ser Ala Thr Pro Ala Leu Tyr Ile Leu  
 100 105 110

Thr Pro Phe Asn Pro Ile Arg Lys Leu Ala Ile Lys Ile Leu Val His  
 115 120 125

Ser Leu Phe Asn Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val  
 130 135 140

Phe Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr  
 145 150 155 160

Thr Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Leu Ala  
 165 170 175

Arg Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn  
 180 185 190

## SSCP Update Sequences.ST25

Trp Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val  
 195 200 205  
 Asp Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala  
 210 215 220  
 Leu Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala  
 225 230 235 240  
 Leu Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val  
 245 250 255  
 Phe Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly  
 260 265 270  
 Asn Leu Arg Asn Lys Cys Leu Gln Trp Pro Pro Asp Asn Ser Ser Phe  
 275 280 285  
 Glu Ile Asn Ile Thr Ser Phe Phe Asn Asn Ser Leu Asp Gly Asn Gly  
 290 295 300  
 Thr Thr Phe Asn Arg Thr Val Ser Ile Phe Asn Trp Asp Glu Tyr Ile  
 305 310 315 320  
 Glu Asp Lys Ser His Phe Tyr Phe Leu Glu Gly Gln Asn Asp Ala Leu  
 325 330 335  
 Leu Cys Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Ile  
 340 345 350  
 Cys Val Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp  
 355 360 365  
 Thr Phe Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp  
 370 375 380  
 Phe Trp Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr  
 385 390 395 400  
 Tyr Met Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu  
 405 410 415  
 Ile Asn Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn  
 420 425 430  
 Gln Ala Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln  
 435 440 445  
 Met Leu Glu Gln Leu Lys Lys Gln Gln Glu Glu Ala Gln Ala Ala Ala  
 450 455 460

## SSCP Update Sequences.ST25

Ala Ala Ala Ser Ala Glu Ser Arg Asp Phe Ser Gly Ala Gly Gly Ile  
 465 470 475 480  
 Gly Val Phe Ser Glu Ser Ser Ser Val Ala Ser Lys Leu Ser Ser Lys  
 485 490 495  
 Ser Glu Lys Glu Leu Lys Asn Arg Arg Lys Lys Lys Lys Gln Lys Glu  
 500 505 510  
 Gln Ser Gly Glu Glu Glu Lys Asn Asp Arg Val Leu Lys Ser Glu Ser  
 515 520 525  
 Glu Asp Ser Ile Arg Arg Lys Gly Phe Arg Phe Ser Leu Glu Gly Ser  
 530 535 540  
 Arg Leu Thr Tyr Glu Lys Arg Phe Ser Ser Pro His Gln Ser Leu Leu  
 545 550 555 560  
 Ser Ile Arg Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Ala Ser  
 565 570 575  
 Leu Phe Ser Phe Arg Gly Arg Ala Lys Asp Ile Gly Ser Glu Asn Asp  
 580 585 590  
 Phe Ala Asp Asp Glu His Ser Thr Phe Glu Asp Asn Asp Ser Arg Arg  
 595 600 605  
 Asp Ser Leu Phe Val Pro His Arg His Gly Glu Arg Arg His Ser Asn  
 610 615 620  
 Val Ser Gln Ala Ser Arg Ala Ser Arg Val Leu Pro Ile Leu Pro Met  
 625 630 635 640  
 Asn Gly Lys Met His Ser Ala Val Asp Cys Asn Gly Val Val Ser Leu  
 645 650 655  
 Val Gly Gly Pro Ser Thr Leu Thr Ser Ala Gly Gln Leu Leu Pro Glu  
 660 665 670  
 Gly Thr Thr Thr Glu Thr Glu Ile Arg Lys Arg Arg Ser Ser Ser Tyr  
 675 680 685  
 His Val Ser Met Asp Leu Leu Glu Asp Pro Thr Ser Arg Gln Arg Ala  
 690 695 700  
 Met Ser Ile Ala Ser Ile Leu Thr Asn Thr Met Glu Glu Leu Glu Glu  
 705 710 715 720  
 Ser Arg Gln Lys Cys Pro Pro Cys Trp Tyr Lys Phe Ala Asn Met Cys  
 725 730 735

## SSCP Update Sequences.ST25

Leu Ile Trp Asp Cys Cys Lys Pro Trp Leu Lys Val Lys His Leu Val  
 740 745 750  
 Asn Leu Val Val Met Asp Pro Phe Val Asp Leu Ala Ile Thr Ile Cys  
 755 760 765  
 Ile Val Leu Asn Thr Leu Phe Met Ala Met Glu His Tyr Pro Met Thr  
 770 775 780  
 Glu Gln Phe Ser Ser Val Leu Ser Val Gly Asn Leu Val Phe Thr Gly  
 785 790 795 800  
 Ile Phe Thr Ala Glu Met Phe Leu Lys Ile Ile Ala Met Asp Pro Tyr  
 805 810 815  
 Tyr Tyr Phe Gln Glu Gly Trp Asn Ile Phe Asp Gly Phe Ile Val Ser  
 820 825 830  
 Leu Ser Leu Met Glu Leu Gly Leu Ala Asn Val Glu Gly Leu Ser Val  
 835 840 845  
 Leu Arg Ser Phe Arg Leu Leu Arg Val Phe Lys Leu Ala Lys Ser Trp  
 850 855 860  
 Pro Thr Leu Asn Met Leu Ile Lys Ile Ile Gly Asn Ser Val Gly Ala  
 865 870 875 880  
 Leu Gly Asn Leu Thr Leu Val Leu Ala Ile Ile Val Phe Ile Phe Ala  
 885 890 895  
 Val Val Gly Met Gln Leu Phe Gly Lys Ser Tyr Lys Glu Cys Val Cys  
 900 905 910  
 Lys Ile Ser Asn Asp Cys Glu Leu Pro Arg Trp His Met His Asp Phe  
 915 920 925  
 Phe His Ser Phe Leu Ile Val Phe Arg Val Leu Cys Gly Glu Trp Ile  
 930 935 940  
 Glu Thr Met Trp Asp Cys Met Glu Val Ala Gly Gln Thr Met Cys Leu  
 945 950 955 960  
 Thr Val Phe Met Met Val Met Val Ile Gly Asn Leu Val Val Leu Asn  
 965 970 975  
 Leu Phe Leu Ala Leu Leu Leu Ser Ser Phe Ser Ser Asp Asn Leu Ala  
 980 985 990  
 Ala Thr Asp Asp Asp Asn Glu Met Asn Asn Ile Gln Ile Ala Val Gly  
 995 1000 1005

## SSCP Update Sequences.ST25

Arg	Met	Gln	Lys	Gly	Ile	Asp	Phe	Val	Lys	Arg	Lys	Ile	Arg	Glu
	1010					1015					1020			
Phe	Ile	Gln	Lys	Ala	Phe	Val	Arg	Lys	Gln	Lys	Ala	Leu	Asp	Glu
	1025					1030					1035			
Ile	Lys	Pro	Leu	Glu	Asp	Leu	Asn	Asn	Lys	Lys	Asp	Ser	Cys	Ile
	1040					1045					1050			
Ser	Asn	His	Thr	Thr	Ile	Glu	Ile	Gly	Lys	Asp	Leu	Asn	Tyr	Leu
	1055					1060					1065			
Lys	Asp	Gly	Asn	Gly	Thr	Thr	Ser	Gly	Ile	Gly	Ser	Ser	Val	Glu
	1070					1075					1080			
Lys	Tyr	Val	Val	Asp	Glu	Ser	Asp	Tyr	Met	Ser	Phe	Ile	Asn	Asn
	1085					1090					1095			
Pro	Ser	Leu	Thr	Val	Thr	Val	Pro	Ile	Ala	Val	Gly	Glu	Ser	Asp
	1100					1105					1110			
Phe	Glu	Asn	Leu	Asn	Thr	Glu	Glu	Phe	Ser	Ser	Glu	Ser	Asp	Met
	1115					1120					1125			
Glu	Glu	Ser	Lys	Glu	Lys	Leu	Asn	Ala	Thr	Ser	Ser	Ser	Glu	Gly
	1130					1135					1140			
Ser	Thr	Val	Asp	Ile	Gly	Ala	Pro	Ala	Glu	Gly	Glu	Gln	Pro	Glu
	1145					1150					1155			
Val	Glu	Pro	Glu	Glu	Ser	Leu	Glu	Pro	Glu	Ala	Cys	Phe	Thr	Glu
	1160					1165					1170			
Asp	Cys	Val	Arg	Lys	Phe	Lys	Cys	Cys	Gln	Ile	Ser	Ile	Glu	Glu
	1175					1180					1185			
Gly	Lys	Gly	Lys	Leu	Trp	Trp	Asn	Leu	Arg	Lys	Thr	Cys	Tyr	Lys
	1190					1195					1200			
Ile	Val	Glu	His	Asn	Trp	Phe	Glu	Thr	Phe	Ile	Val	Phe	Met	Ile
	1205					1210					1215			
Leu	Leu	Ser	Ser	Gly	Ala	Leu	Ala	Phe	Glu	Asp	Ile	Tyr	Ile	Glu
	1220					1225					1230			
Gln	Arg	Lys	Thr	Ile	Lys	Thr	Met	Leu	Glu	Tyr	Ala	Asp	Lys	Val
	1235					1240					1245			
Phe	Thr	Tyr	Ile	Phe	Ile	Leu	Glu	Met	Leu	Leu	Lys	Trp	Val	Ala
	1250					1255					1260			

## SSCP Update Sequences.ST25

Tyr Gly Phe Gln Val Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp  
 1265 1270 1275  
 Phe Leu Ile Val Asp Val Ser Leu Val Ser Leu Thr Ala Asn Ala  
 1280 1285 1290  
 Leu Gly Tyr Ser Glu Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu  
 1295 1300 1305  
 Arg Ala Leu Arg Pro Leu Arg Ala Leu Ser Arg Phe Glu Gly Met  
 1310 1315 1320  
 Arg Val Val Val Asn Ala Leu Leu Gly Ala Ile Pro Ser Ile Met  
 1325 1330 1335  
 Asn Val Leu Leu Val Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile  
 1340 1345 1350  
 Met Gly Val Asn Leu Phe Ala Gly Lys Phe Tyr His Cys Ile Asn  
 1355 1360 1365  
 Tyr Thr Thr Gly Glu Met Phe Asp Val Ser Val Val Asn Asn Tyr  
 1370 1375 1380  
 Ser Glu Cys Lys Ala Leu Ile Glu Ser Asn Gln Thr Ala Arg Trp  
 1385 1390 1395  
 Lys Asn Val Lys Val Asn Phe Asp Asn Val Gly Leu Gly Tyr Leu  
 1400 1405 1410  
 Ser Leu Leu Gln Val Ala Thr Phe Lys Gly Trp Met Asp Ile Met  
 1415 1420 1425  
 Tyr Ala Ala Val Asp Ser Arg Asn Val Glu Leu Gln Pro Lys Tyr  
 1430 1435 1440  
 Glu Asp Asn Leu Tyr Met Tyr Leu Tyr Phe Val Ile Phe Ile Ile  
 1445 1450 1455  
 Phe Gly Ser Phe Phe Thr Leu Asn Leu Phe Ile Gly Val Ile Ile  
 1460 1465 1470  
 Asp Asn Phe Asn Gln Gln Lys Lys Lys Phe Gly Gly Gln Asp Ile  
 1475 1480 1485  
 Phe Met Thr Glu Glu Gln Lys Lys Tyr Tyr Asn Ala Met Lys Lys  
 1490 1495 1500  
 Leu Gly Ser Lys Lys Pro Gln Lys Pro Ile Pro Arg Pro Ala Asn  
 1505 1510 1515

## SSCP Update Sequences.ST25

Lys	Phe	Gln	Gly	Met	Val	Phe	Asp	Phe	Val	Thr	Lys	Gln	Val	Phe
	1520					1525					1530			
Asp	Ile	Ser	Ile	Met	Ile	Leu	Ile	Cys	Leu	Asn	Met	Val	Thr	Met
	1535					1540					1545			
Met	Val	Glu	Thr	Asp	Asp	Gln	Ser	Gln	Glu	Met	Thr	Asn	Ile	Leu
	1550					1555					1560			
Tyr	Trp	Ile	Asn	Leu	Val	Phe	Ile	Val	Leu	Phe	Thr	Gly	Glu	Cys
	1565					1570					1575			
Val	Leu	Lys	Leu	Ile	Ser	Leu	Arg	Tyr	Tyr	Tyr	Phe	Thr	Ile	Gly
	1580					1585					1590			
Trp	Asn	Ile	Phe	Asp	Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly
	1595					1600					1605			
Met	Phe	Leu	Ala	Glu	Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr
	1610					1615					1620			
Leu	Phe	Arg	Val	Ile	Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg
	1625					1630					1635			
Leu	Ile	Lys	Gly	Ala	Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu
	1640					1645					1650			
Met	Met	Ser	Leu	Pro	Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe
	1655					1660					1665			
Leu	Val	Met	Phe	Ile	Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala
	1670					1675					1680			
Tyr	Val	Lys	Arg	Glu	Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu
	1685					1690					1695			
Thr	Phe	Gly	Asn	Ser	Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser
	1700					1705					1710			
Ala	Gly	Trp	Asp	Gly	Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Gly	Pro
	1715					1720					1725			
Pro	Asp	Cys	Asp	Pro	Asp	Lys	Asp	His	Pro	Gly	Ser	Ser	Val	Lys
	1730					1735					1740			
Gly	Asp	Cys	Gly	Asn	Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser
	1745					1750					1755			
Tyr	Ile	Ile	Ile	Ser	Phe	Leu	Val	Val	Leu	Asn	Met	Tyr	Ile	Ala
	1760					1765					1770			



## SSCP Update Sequences.ST25

Val Ile Leu Glu Asn Phe Ser Val Ala Thr Glu Glu Ser Ala Glu  
 1775 1780 1785  
 Pro Leu Ser Glu Asp Asp Phe Glu Met Phe Tyr Glu Val Trp Glu  
 1790 1795 1800  
 Lys Phe Asp Pro Asp Ala Thr Gln Phe Ile Glu Phe Ala Lys Leu  
 1805 1810 1815  
 Ser Asp Phe Ala Asp Ala Leu Asp Pro Pro Leu Leu Ile Ala Lys  
 1820 1825 1830  
 Pro Asn Lys Val Gln Leu Ile Ala Met Asp Leu Pro Met Val Ser  
 1835 1840 1845  
 Gly Asp Arg Ile His Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys  
 1850 1855 1860  
 Arg Val Leu Gly Glu Ser Gly Glu Met Asp Ala Leu Arg Ile Gln  
 1865 1870 1875  
 Met Glu Glu Arg Phe Met Ala Ser Asn Pro Ser Lys Val Ser Tyr  
 1880 1885 1890  
 Glu Pro Ile Thr Thr Thr Leu Lys Arg Lys Gln Glu Glu Val Ser  
 1895 1900 1905  
 Ala Ile Ile Ile Gln Arg Ala Tyr Arg Arg Tyr Leu Leu Lys Gln  
 1910 1915 1920  
 Lys Val Lys Lys Val Ser Ser Ile Tyr Lys Lys Asp Lys Gly Lys  
 1925 1930 1935  
 Glu Cys Asp Gly Thr Pro Ile Lys Glu Asp Thr Leu Ile Asp Lys  
 1940 1945 1950  
 Leu Asn Glu Asn Ser Thr Pro Glu Lys Thr Asp Met Thr Pro Ser  
 1955 1960 1965  
 Thr Thr Ser Pro Pro Ser Tyr Asp Ser Val Thr Lys Pro Glu Lys  
 1970 1975 1980  
 Glu Lys Phe Glu Lys Asp Lys Ser Glu Lys Glu Asp Lys Gly Lys  
 1985 1990 1995  
 Asp Ile Arg Glu Ser Lys Lys  
 2000 2005

<210> 86  
 <211> 2005  
 <212> PRT

## SSCP Update Sequences.ST25

&lt;213&gt; Homo sapiens

&lt;400&gt; 86

Met Ala Gln Ser Val Leu Val Pro Pro Gly Pro Asp Ser Phe Arg Phe  
 1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Gln Arg Ile Ala Glu Glu  
 20 25 30

Lys Ala Lys Arg Pro Lys Gln Glu Arg Lys Asp Glu Asp Asp Glu Asn  
 35 40 45

Gly Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Ser Leu Pro Phe  
 50 55 60

Ile Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Val Pro Leu Glu Asp  
 65 70 75 80

Leu Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys  
 85 90 95

Gly Lys Ala Ile Ser Arg Phe Ser Ala Thr Pro Ala Leu Tyr Ile Leu  
 100 105 110

Thr Pro Phe Asn Pro Ile Arg Lys Leu Ala Ile Lys Ile Leu Val His  
 115 120 125

Ser Leu Phe Asn Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val  
 130 135 140

Phe Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr  
 145 150 155 160

Thr Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Leu Ala  
 165 170 175

Arg Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn  
 180 185 190

Trp Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val  
 195 200 205

Asp Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala  
 210 215 220

Leu Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala  
 225 230 235 240

Leu Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val  
 245 250 255

## SSCP Update Sequences.ST25

Phe Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly  
 260 265 270  
 Asn Leu Arg Asn Lys Cys Leu Gln Trp Pro Pro Asp Asn Ser Ser Phe  
 275 280 285  
 Glu Ile Asn Ile Thr Ser Phe Phe Asn Asn Ser Leu Asp Gly Asn Gly  
 290 295 300  
 Thr Thr Phe Asn Arg Thr Val Ser Ile Phe Asn Trp Asp Glu Tyr Ile  
 305 310 315 320  
 Glu Asp Lys Ser His Phe Tyr Phe Leu Glu Gly Gln Asn Asp Ala Leu  
 325 330 335  
 Leu Cys Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Ile  
 340 345 350  
 Cys Val Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp  
 355 360 365  
 Thr Phe Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp  
 370 375 380  
 Phe Trp Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr  
 385 390 395 400  
 Tyr Met Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu  
 405 410 415  
 Ile Asn Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn  
 420 425 430  
 Gln Ala Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln  
 435 440 445  
 Met Leu Glu Gln Leu Lys Lys Gln Gln Glu Glu Ala Gln Ala Ala Ala  
 450 455 460  
 Ala Ala Ala Ser Ala Glu Ser Arg Asp Phe Ser Gly Ala Gly Gly Ile  
 465 470 475 480  
 Gly Val Phe Ser Glu Ser Ser Ser Val Ala Ser Lys Leu Ser Ser Lys  
 485 490 495  
 Ser Glu Lys Glu Leu Lys Asn Arg Arg Lys Lys Lys Lys Gln Lys Glu  
 500 505 510  
 Gln Ser Gly Glu Glu Glu Lys Asn Asp Arg Val Leu Lys Ser Glu Ser  
 515 520 525

## SSCP Update Sequences.ST25

Glu Asp Ser Ile Arg Arg Lys Gly Phe Arg Phe Ser Leu Glu Gly Ser  
 530 535 540  
 Arg Leu Thr Tyr Glu Lys Arg Phe Ser Ser Pro His Gln Ser Leu Leu  
 545 550 555  
 Ser Ile Arg Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Ala Ser  
 565 570 575  
 Leu Phe Ser Phe Arg Gly Arg Ala Lys Asp Ile Gly Ser Glu Asn Asp  
 580 585 590  
 Phe Ala Asp Asp Glu His Ser Thr Phe Glu Asp Asn Asp Ser Arg Arg  
 595 600 605  
 Asp Ser Leu Phe Val Pro His Arg His Gly Glu Arg Arg His Ser Asn  
 610 615 620  
 Val Ser Gln Ala Ser Arg Ala Ser Arg Val Leu Pro Ile Leu Pro Met  
 625 630 635 640  
 Asn Gly Lys Met His Ser Ala Val Asp Cys Asn Gly Val Val Ser Leu  
 645 650 655  
 Val Gly Gly Pro Ser Thr Leu Thr Ser Ala Gly Gln Leu Leu Pro Glu  
 660 665 670  
 Gly Thr Thr Thr Glu Thr Glu Ile Arg Lys Arg Arg Ser Ser Ser Tyr  
 675 680 685  
 His Val Ser Met Asp Leu Leu Glu Asp Pro Thr Ser Arg Gln Arg Ala  
 690 695 700  
 Met Ser Ile Ala Ser Ile Leu Thr Asn Thr Met Glu Glu Leu Glu Glu  
 705 710 715 720  
 Ser Arg Gln Lys Cys Pro Pro Cys Trp Tyr Lys Phe Ala Asn Met Cys  
 725 730 735  
 Leu Ile Trp Asp Cys Cys Lys Pro Trp Leu Lys Val Lys His Leu Val  
 740 745 750  
 Asn Leu Val Val Met Asp Pro Phe Val Asp Leu Ala Ile Thr Ile Cys  
 755 760 765  
 Ile Val Leu Asn Thr Leu Phe Met Ala Met Glu His Tyr Pro Met Thr  
 770 775 780  
 Glu Gln Phe Ser Ser Val Leu Ser Val Gly Asn Leu Val Phe Thr Gly  
 785 790 795 800

## SSCP Update Sequences.ST25

Ile Phe Thr Ala Glu Met Phe Leu Lys Ile Ile Ala Met Asp Pro Tyr  
 805 810 815  
 Tyr Tyr Phe Gln Glu Gly Trp Asn Ile Phe Asp Gly Phe Ile Val Ser  
 820 825 830  
 Leu Ser Leu Met Glu Leu Gly Leu Ala Asn Val Glu Gly Leu Ser Val  
 835 840 845  
 Leu Arg Ser Phe Arg Leu Leu Arg Val Phe Lys Leu Ala Lys Ser Trp  
 850 855 860  
 Pro Thr Leu Asn Met Leu Ile Lys Ile Ile Gly Asn Ser Val Gly Ala  
 865 870 875 880  
 Leu Gly Asn Leu Thr Leu Val Leu Ala Ile Ile Val Phe Ile Phe Ala  
 885 890 895  
 Val Val Gly Met Gln Leu Phe Gly Lys Ser Tyr Lys Glu Cys Val Cys  
 900 905 910  
 Lys Ile Ser Asn Asp Cys Glu Leu Pro Arg Trp His Met His Asp Phe  
 915 920 925  
 Phe His Ser Phe Leu Ile Val Phe Arg Val Leu Cys Gly Glu Trp Ile  
 930 935 940  
 Glu Thr Met Trp Asp Cys Met Glu Val Ala Gly Gln Thr Met Cys Leu  
 945 950 955 960  
 Thr Val Phe Met Met Val Met Val Ile Gly Asn Leu Val Val Leu Asn  
 965 970 975  
 Leu Phe Leu Ala Leu Leu Leu Ser Ser Phe Ser Ser Asp Asn Leu Ala  
 980 985 990  
 Ala Thr Asp Asp Asp Asn Glu Met Asn Asn Leu Gln Ile Ala Val Gly  
 995 1000 1005  
 Arg Met Gln Lys Gly Ile Asp Phe Val Lys Arg Lys Ile Arg Glu  
 1010 1015 1020  
 Phe Ile Gln Lys Ala Phe Val Arg Lys Gln Lys Ala Leu Asp Glu  
 1025 1030 1035  
 Ile Lys Pro Leu Glu Asp Leu Asn Asn Lys Lys Asp Ser Cys Ile  
 1040 1045 1050  
 Ser Asn His Thr Thr Ile Glu Ile Gly Lys Asp Leu Asn Tyr Leu  
 1055 1060 1065

## SSCP Update Sequences.ST25

Lys Asp Gly Asn Gly Thr Thr Ser Gly Ile Gly Ser Ser Val Glu  
 1070 1075 1080  
 Lys Tyr Val Val Asp Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn  
 1085 1090 1095  
 Pro Ser Leu Thr Val Thr Val Pro Ile Ala Val Gly Glu Ser Asp  
 1100 1105 1110  
 Phe Glu Asn Leu Asn Thr Glu Glu Phe Ser Ser Glu Ser Asp Met  
 1115 1120 1125  
 Glu Glu Ser Lys Glu Lys Leu Asn Ala Thr Ser Ser Ser Glu Gly  
 1130 1135 1140  
 Ser Thr Val Asp Ile Gly Ala Pro Ala Glu Gly Glu Gln Pro Glu  
 1145 1150 1155  
 Val Glu Pro Glu Glu Ser Leu Glu Pro Glu Ala Cys Phe Thr Glu  
 1160 1165 1170  
 Asp Cys Val Arg Lys Phe Lys Cys Cys Gln Ile Ser Ile Glu Glu  
 1175 1180 1185  
 Gly Lys Gly Lys Leu Trp Trp Asn Leu Arg Lys Ala Cys Tyr Lys  
 1190 1195 1200  
 Ile Val Glu His Asn Trp Phe Glu Thr Phe Ile Val Phe Met Ile  
 1205 1210 1215  
 Leu Leu Ser Ser Gly Ala Leu Ala Phe Glu Asp Ile Tyr Ile Glu  
 1220 1225 1230  
 Gln Arg Lys Thr Ile Lys Thr Met Leu Glu Tyr Ala Asp Lys Val  
 1235 1240 1245  
 Phe Thr Tyr Ile Phe Ile Leu Glu Met Leu Leu Lys Trp Val Ala  
 1250 1255 1260  
 Tyr Gly Phe Gln Val Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp  
 1265 1270 1275  
 Phe Leu Ile Val Asp Val Ser Leu Val Ser Leu Thr Ala Asn Ala  
 1280 1285 1290  
 Leu Gly Tyr Ser Glu Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu  
 1295 1300 1305  
 Arg Ala Leu Arg Pro Leu Arg Ala Leu Ser Arg Phe Glu Gly Met  
 1310 1315 1320

## SSCP Update Sequences.ST25

Arg Val Val Val Asn Ala Leu Leu Gly Ala Ile Pro Ser Ile Met  
 1325 1330 1335  
 Asn Val Leu Leu Val Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile  
 1340 1345 1350  
 Met Gly Val Asn Leu Phe Ala Gly Lys Phe Tyr His Cys Ile Asn  
 1355 1360 1365  
 Tyr Thr Thr Gly Glu Met Phe Asp Val Ser Val Val Asn Asn Tyr  
 1370 1375 1380  
 Ser Glu Cys Lys Ala Leu Ile Glu Ser Asn Gln Thr Ala Arg Trp  
 1385 1390 1395  
 Lys Asn Val Lys Val Asn Phe Asp Asn Val Gly Leu Gly Tyr Leu  
 1400 1405 1410  
 Ser Leu Leu Gln Val Ala Thr Phe Lys Gly Trp Met Asp Ile Met  
 1415 1420 1425  
 Tyr Ala Ala Val Asp Ser Arg Asn Val Glu Leu Gln Pro Lys Tyr  
 1430 1435 1440  
 Glu Asp Asn Leu Tyr Met Tyr Leu Tyr Phe Val Ile Phe Ile Ile  
 1445 1450 1455  
 Phe Gly Ser Phe Phe Thr Leu Asn Leu Phe Ile Gly Val Ile Ile  
 1460 1465 1470  
 Asp Asn Phe Asn Gln Gln Lys Lys Lys Phe Gly Gly Gln Asp Ile  
 1475 1480 1485  
 Phe Met Thr Glu Glu Gln Lys Lys Tyr Tyr Asn Ala Met Lys Lys  
 1490 1495 1500  
 Leu Gly Ser Lys Lys Pro Gln Lys Pro Ile Pro Arg Pro Ala Asn  
 1505 1510 1515  
 Lys Phe Gln Gly Met Val Phe Asp Phe Val Thr Lys Gln Val Phe  
 1520 1525 1530  
 Asp Ile Ser Ile Met Ile Leu Ile Cys Leu Asn Met Val Thr Met  
 1535 1540 1545  
 Met Val Glu Thr Asp Asp Gln Ser Gln Glu Met Thr Asn Ile Leu  
 1550 1555 1560  
 Tyr Trp Ile Asn Leu Val Phe Ile Val Leu Phe Thr Gly Glu Cys  
 1565 1570 1575

## SSCP Update Sequences.ST25

Val	Leu	Lys	Leu	Ile	Ser	Leu	Arg	Tyr	Tyr	Tyr	Phe	Thr	Ile	Gly
	1580					1585					1590			
Trp	Asn	Ile	Phe	Asp	Phe	Val	Val	Val	Ile	Leu	Ser	Ile	Val	Gly
	1595					1600					1605			
Met	Phe	Leu	Ala	Glu	Leu	Ile	Glu	Lys	Tyr	Phe	Val	Ser	Pro	Thr
	1610					1615					1620			
Leu	Phe	Arg	Val	Ile	Arg	Leu	Ala	Arg	Ile	Gly	Arg	Ile	Leu	Arg
	1625					1630					1635			
Leu	Ile	Lys	Gly	Ala	Lys	Gly	Ile	Arg	Thr	Leu	Leu	Phe	Ala	Leu
	1640					1645					1650			
Met	Met	Ser	Leu	Pro	Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe
	1655					1660					1665			
Leu	Val	Met	Phe	Ile	Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala
	1670					1675					1680			
Tyr	Val	Lys	Arg	Glu	Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu
	1685					1690					1695			
Thr	Phe	Gly	Asn	Ser	Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser
	1700					1705					1710			
Ala	Gly	Trp	Asp	Gly	Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Gly	Pro
	1715					1720					1725			
Pro	Asp	Cys	Asp	Pro	Asp	Lys	Asp	His	Pro	Gly	Ser	Ser	Val	Lys
	1730					1735					1740			
Gly	Asp	Cys	Gly	Asn	Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser
	1745					1750					1755			
Tyr	Ile	Ile	Ile	Ser	Phe	Leu	Val	Val	Leu	Asn	Met	Tyr	Ile	Ala
	1760					1765					1770			
Val	Ile	Leu	Glu	Asn	Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu
	1775					1780					1785			
Pro	Leu	Ser	Glu	Asp	Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu
	1790					1795					1800			
Lys	Phe	Asp	Pro	Asp	Ala	Thr	Gln	Phe	Ile	Glu	Phe	Ala	Lys	Leu
	1805					1810					1815			
Ser	Asp	Phe	Ala	Asp	Ala	Leu	Asp	Pro	Pro	Leu	Leu	Ile	Ala	Lys
	1820					1825					1830			



## SSCP Update Sequences.ST25

Pro Asn Lys Val Gln Leu Ile Ala Met Asp Leu Pro Met Val Ser  
 1835 1840 1845

Gly Asp Arg Ile His Cys Leu Asp Ile Leu Phe Ala Phe Thr Lys  
 1850 1855 1860

Arg Val Leu Gly Glu Ser Gly Glu Met Asp Ala Leu Arg Ile Gln  
 1865 1870 1875

Met Glu Glu Arg Phe Met Ala Ser Asn Pro Ser Lys Val Ser Tyr  
 1880 1885 1890

Glu Pro Ile Thr Thr Thr Leu Lys Arg Lys Gln Glu Glu Val Ser  
 1895 1900 1905

Ala Ile Ile Ile Gln Arg Ala Tyr Arg Arg Tyr Leu Leu Lys Gln  
 1910 1915 1920

Lys Val Lys Lys Val Ser Ser Ile Tyr Lys Lys Asp Lys Gly Lys  
 1925 1930 1935

Glu Cys Asp Gly Thr Pro Ile Lys Glu Asp Thr Leu Ile Asp Lys  
 1940 1945 1950

Leu Asn Glu Asn Ser Thr Pro Glu Lys Thr Asp Met Thr Pro Ser  
 1955 1960 1965

Thr Thr Ser Pro Pro Ser Tyr Asp Ser Val Thr Lys Pro Glu Lys  
 1970 1975 1980

Glu Lys Phe Glu Lys Asp Lys Ser Glu Lys Glu Asp Lys Gly Lys  
 1985 1990 1995

Asp Ile Arg Glu Ser Lys Lys  
 2000 2005

<210> 87  
 <211> 2005  
 <212> PRT  
 <213> Homo sapiens

<400> 87

Met Ala Gln Ser Val Leu Val Pro Pro Gly Pro Asp Ser Phe Arg Phe  
 1 5 10 15

Phe Thr Arg Glu Ser Leu Ala Ala Ile Glu Gln Arg Ile Ala Glu Glu  
 20 25 30

Lys Ala Lys Arg Pro Lys Gln Glu Arg Lys Asp Glu Asp Asp Glu Asn  
 35 40 45

Gly Pro Lys Pro Asn Ser Asp Leu Glu Ala Gly Lys Ser Leu Pro Phe

## SSCP Update Sequences.ST25

50

55

60

Ile Tyr Gly Asp Ile Pro Pro Glu Met Val Ser Val Pro Leu Glu Asp  
65 70 75 80

Leu Asp Pro Tyr Tyr Ile Asn Lys Lys Thr Phe Ile Val Leu Asn Lys  
85 90 95

Gly Lys Ala Ile Ser Arg Phe Ser Ala Thr Pro Ala Leu Tyr Ile Leu  
100 105 110

Thr Pro Phe Asn Pro Ile Arg Lys Leu Ala Ile Lys Ile Leu Val His  
115 120 125

Ser Leu Phe Asn Met Leu Ile Met Cys Thr Ile Leu Thr Asn Cys Val  
130 135 140

Phe Met Thr Met Ser Asn Pro Pro Asp Trp Thr Lys Asn Val Glu Tyr  
145 150 155 160

Thr Phe Thr Gly Ile Tyr Thr Phe Glu Ser Leu Ile Lys Ile Leu Ala  
165 170 175

Arg Gly Phe Cys Leu Glu Asp Phe Thr Phe Leu Arg Asp Pro Trp Asn  
180 185 190

Trp Leu Asp Phe Thr Val Ile Thr Phe Ala Tyr Val Thr Glu Phe Val  
195 200 205

Asp Leu Gly Asn Val Ser Ala Leu Arg Thr Phe Arg Val Leu Arg Ala  
210 215 220

Leu Lys Thr Ile Ser Val Ile Pro Gly Leu Lys Thr Ile Val Gly Ala  
225 230 235 240

Leu Ile Gln Ser Val Lys Lys Leu Ser Asp Val Met Ile Leu Thr Val  
245 250 255

Phe Cys Leu Ser Val Phe Ala Leu Ile Gly Leu Gln Leu Phe Met Gly  
260 265 270

Asn Leu Arg Asn Lys Cys Leu Gln Trp Pro Pro Asp Asn Ser Ser Phe  
275 280 285

Glu Ile Asn Ile Thr Ser Phe Phe Asn Asn Ser Leu Asp Gly Asn Gly  
290 295 300

Thr Thr Phe Asn Arg Thr Val Ser Ile Phe Asn Trp Asp Glu Tyr Ile  
305 310 315 320

Glu Asp Lys Ser His Phe Tyr Phe Leu Glu Gly Gln Asn Asp Ala Leu

## SSCP Update Sequences.ST25

325

330

335

Leu Cys Gly Asn Ser Ser Asp Ala Gly Gln Cys Pro Glu Gly Tyr Ile  
 340 345 350

Cys Val Lys Ala Gly Arg Asn Pro Asn Tyr Gly Tyr Thr Ser Phe Asp  
 355 360 365

Thr Phe Ser Trp Ala Phe Leu Ser Leu Phe Arg Leu Met Thr Gln Asp  
 370 375 380

Phe Trp Glu Asn Leu Tyr Gln Leu Thr Leu Arg Ala Ala Gly Lys Thr  
 385 390 395 400

Tyr Met Ile Phe Phe Val Leu Val Ile Phe Leu Gly Ser Phe Tyr Leu  
 405 410 415

Ile Asn Leu Ile Leu Ala Val Val Ala Met Ala Tyr Glu Glu Gln Asn  
 420 425 430

Gln Ala Thr Leu Glu Glu Ala Glu Gln Lys Glu Ala Glu Phe Gln Gln  
 435 440 445

Met Leu Glu Gln Leu Lys Lys Gln Gln Glu Glu Ala Gln Ala Ala Ala  
 450 455 460

Ala Ala Ala Ser Ala Glu Ser Arg Asp Phe Ser Gly Ala Gly Gly Ile  
 465 470 475 480

Gly Val Phe Ser Glu Ser Ser Ser Val Ala Ser Lys Leu Ser Ser Lys  
 485 490 495

Ser Glu Lys Glu Leu Lys Asn Arg Arg Lys Lys Lys Lys Gln Lys Glu  
 500 505 510

Gln Ser Gly Glu Glu Glu Lys Asn Asp Arg Val Leu Lys Ser Glu Ser  
 515 520 525

Glu Asp Ser Ile Arg Arg Lys Gly Phe Arg Phe Ser Leu Glu Gly Ser  
 530 535 540

Arg Leu Thr Tyr Glu Lys Arg Phe Ser Ser Pro His Gln Ser Leu Leu  
 545 550 555 560

Ser Ile Arg Gly Ser Leu Phe Ser Pro Arg Arg Asn Ser Arg Ala Ser  
 565 570 575

Leu Phe Ser Phe Arg Gly Arg Ala Lys Asp Ile Gly Ser Glu Asn Asp  
 580 585 590

Phe Ala Asp Asp Glu His Ser Thr Phe Glu Asp Asn Asp Ser Arg Arg  
 Page 209

SSCP Update Sequences.ST25

595 600 605

Asp Ser Leu Phe Val Pro His Arg His Gly Glu Arg Arg His Ser Asn  
610 615 620

Val Ser Gln Ala Ser Arg Ala Ser Arg Val Leu Pro Ile Leu Pro Met  
625 630 635 640

Asn Gly Lys Met His Ser Ala Val Asp Cys Asn Gly Val Val Ser Leu  
645 650 655

Val Gly Gly Pro Ser Thr Leu Thr Ser Ala Gly Gln Leu Leu Pro Glu  
660 665 670

Gly Thr Thr Thr Glu Thr Glu Ile Arg Lys Arg Arg Ser Ser Ser Tyr  
675 680 685

His Val Ser Met Asp Leu Leu Glu Asp Pro Thr Ser Arg Gln Arg Ala  
690 695 700

Met Ser Ile Ala Ser Ile Leu Thr Asn Thr Met Glu Glu Leu Glu Glu  
705 710 715 720

Ser Arg Gln Lys Cys Pro Pro Cys Trp Tyr Lys Phe Ala Asn Met Cys  
725 730 735

Leu Ile Trp Asp Cys Cys Lys Pro Trp Leu Lys Val Lys His Leu Val  
740 745 750

Asn Leu Val Val Met Asp Pro Phe Val Asp Leu Ala Ile Thr Ile Cys  
755 760 765

Ile Val Leu Asn Thr Leu Phe Met Ala Met Glu His Tyr Pro Met Thr  
770 775 780

Glu Gln Phe Ser Ser Val Leu Ser Val Gly Asn Leu Val Phe Thr Gly  
785 790 795 800

Ile Phe Thr Ala Glu Met Phe Leu Lys Ile Ile Ala Met Asp Pro Tyr  
805 810 815

Tyr Tyr Phe Gln Glu Gly Trp Asn Ile Phe Asp Gly Phe Ile Val Ser  
820 825 830

Leu Ser Leu Met Glu Leu Gly Leu Ala Asn Val Glu Gly Leu Ser Val  
835 840 845

Leu Arg Ser Phe Arg Leu Leu Arg Val Phe Lys Leu Ala Lys Ser Trp  
850 855 860

Pro Thr Leu Asn Met Leu Ile Lys Ile Ile Gly Asn Ser Val Gly Ala

## SSCP Update Sequences.ST25

865                      870                      875                      880  
 Leu Gly Asn Leu Thr Leu Val Leu Ala Ile Ile Val Phe Ile Phe Ala  
                                  885                      890                      895  
 Val Val Gly Met Gln Leu Phe Gly Lys Ser Tyr Lys Glu Cys Val Cys  
                                  900                      905                      910  
 Lys Ile Ser Asn Asp Cys Glu Leu Pro Arg Trp His Met His Asp Phe  
                                  915                      920                      925  
 Phe His Ser Phe Leu Ile Val Phe Arg Val Leu Cys Gly Glu Trp Ile  
                                  930                      935                      940  
 Glu Thr Met Trp Asp Cys Met Glu Val Ala Gly Gln Thr Met Cys Leu  
                                  945                      950                      955                      960  
 Thr Val Phe Met Met Val Met Val Ile Gly Asn Leu Val Val Leu Asn  
                                  965                      970                      975  
 Leu Phe Leu Ala Leu Leu Leu Ser Ser Phe Ser Ser Asp Asn Leu Ala  
                                  980                      985                      990  
 Ala Thr Asp Asp Asp Asn Glu Met Asn Asn Leu Gln Ile Ala Val Gly  
                                  995                      1000                      1005  
 Arg Met Gln Lys Gly Ile Asp Phe Val Lys Arg Lys Ile Arg Glu  
                                  1010                      1015                      1020  
 Phe Ile Gln Lys Ala Phe Val Arg Lys Gln Lys Ala Leu Asp Glu  
                                  1025                      1030                      1035  
 Ile Lys Pro Leu Glu Asp Leu Asn Asn Lys Lys Asp Ser Cys Ile  
                                  1040                      1045                      1050  
 Ser Asn His Thr Thr Ile Glu Ile Gly Lys Asp Leu Asn Tyr Leu  
                                  1055                      1060                      1065  
 Lys Asp Gly Asn Gly Thr Thr Ser Gly Ile Gly Ser Ser Val Glu  
                                  1070                      1075                      1080  
 Lys Tyr Val Val Asp Glu Ser Asp Tyr Met Ser Phe Ile Asn Asn  
                                  1085                      1090                      1095  
 Pro Ser Leu Thr Val Thr Val Pro Ile Ala Val Gly Glu Ser Asp  
                                  1100                      1105                      1110  
 Phe Glu Asn Leu Asn Thr Glu Glu Phe Ser Ser Glu Ser Asp Met  
                                  1115                      1120                      1125  
 Glu Glu Ser Lys Glu Lys Leu Asn Ala Thr Ser Ser Ser Glu Gly

## SSCP Update Sequences.ST25

1130

1135

1140

Ser Thr Val Asp Ile Gly Ala Pro Ala Glu Gly Glu Gln Pro Glu  
 1145 1150 1155

Val Glu Pro Glu Glu Ser Leu Glu Pro Glu Ala Cys Phe Thr Glu  
 1160 1165 1170

Asp Cys Val Arg Lys Phe Lys Cys Cys Gln Ile Ser Ile Glu Glu  
 1175 1180 1185

Gly Lys Gly Lys Leu Trp Trp Asn Leu Arg Lys Thr Cys Tyr Lys  
 1190 1195 1200

Ile Val Glu His Asn Trp Phe Glu Thr Phe Ile Val Phe Met Ile  
 1205 1210 1215

Leu Leu Ser Ser Gly Ala Leu Ala Phe Glu Asp Ile Tyr Ile Glu  
 1220 1225 1230

Gln Arg Lys Thr Ile Lys Thr Met Leu Glu Tyr Ala Asp Lys Val  
 1235 1240 1245

Phe Thr Tyr Ile Phe Ile Leu Glu Met Leu Leu Lys Trp Val Ala  
 1250 1255 1260

Tyr Gly Phe Gln Val Tyr Phe Thr Asn Ala Trp Cys Trp Leu Asp  
 1265 1270 1275

Phe Leu Ile Val Asp Val Ser Leu Val Ser Leu Thr Ala Asn Ala  
 1280 1285 1290

Leu Gly Tyr Ser Glu Leu Gly Ala Ile Lys Ser Leu Arg Thr Leu  
 1295 1300 1305

Arg Ala Leu Arg Pro Leu Arg Ala Leu Ser Gln Phe Glu Gly Met  
 1310 1315 1320

Arg Val Val Val Asn Ala Leu Leu Gly Ala Ile Pro Ser Ile Met  
 1325 1330 1335

Asn Val Leu Leu Val Cys Leu Ile Phe Trp Leu Ile Phe Ser Ile  
 1340 1345 1350

Met Gly Val Asn Leu Phe Ala Gly Lys Phe Tyr His Cys Ile Asn  
 1355 1360 1365

Tyr Thr Thr Gly Glu Met Phe Asp Val Ser Val Val Asn Asn Tyr  
 1370 1375 1380

Ser Glu Cys Lys Ala Leu Ile Glu Ser Asn Gln Thr Ala Arg Trp

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## SSCP update Sequences ST25

1640														
Met	Met	Ser	Leu	Pro	Ala	Leu	Phe	Asn	Ile	Gly	Leu	Leu	Leu	Phe
1655						1660					1665			
Leu	Val	Met	Phe	Ile	Tyr	Ala	Ile	Phe	Gly	Met	Ser	Asn	Phe	Ala
1670						1675					1680			
Tyr	Val	Lys	Arg	Glu	Val	Gly	Ile	Asp	Asp	Met	Phe	Asn	Phe	Glu
1685						1690					1695			
Thr	Phe	Gly	Asn	Ser	Met	Ile	Cys	Leu	Phe	Gln	Ile	Thr	Thr	Ser
1700						1705					1710			
Ala	Gly	Trp	Asp	Gly	Leu	Leu	Ala	Pro	Ile	Leu	Asn	Ser	Gly	Pro
1715						1720					1725			
Pro	Asp	Cys	Asp	Pro	Asp	Lys	Asp	His	Pro	Gly	Ser	Ser	Val	Lys
1730						1735					1740			
Gly	Asp	Cys	Gly	Asn	Pro	Ser	Val	Gly	Ile	Phe	Phe	Phe	Val	Ser
1745						1750					1755			
Tyr	Ile	Ile	Ile	Ser	Phe	Leu	Val	Val	Leu	Asn	Met	Tyr	Ile	Ala
1760						1765					1770			
Val	Ile	Leu	Glu	Asn	Phe	Ser	Val	Ala	Thr	Glu	Glu	Ser	Ala	Glu
1775						1780					1785			
Pro	Leu	Ser	Glu	Asp	Asp	Phe	Glu	Met	Phe	Tyr	Glu	Val	Trp	Glu
1790						1795					1800			
Lys	Phe	Asp	Pro	Asp	Ala	Thr	Gln	Phe	Ile	Glu	Phe	Ala	Lys	Leu
1805						1810					1815			
Ser	Asp	Phe	Ala	Asp	Ala	Leu	Asp	Pro	Pro	Leu	Leu	Ile	Ala	Lys
1820						1825					1830			
Pro	Asn	Lys	Val	Gln	Leu	Ile	Ala	Met	Asp	Leu	Pro	Met	Val	Ser
1835						1840					1845			
Gly	Asp	Arg	Ile	His	Cys	Leu	Asp	Ile	Leu	Phe	Ala	Phe	Thr	Lys
1850						1855					1860			
Arg	Val	Leu	Gly	Glu	Ser	Gly	Glu	Met	Asp	Ala	Leu	Arg	Ile	Gln
1865						1870					1875			
Met	Glu	Glu	Arg	Phe	Met	Ala	Ser	Asn	Pro	Ser	Lys	Val	Ser	Tyr
1880						1885					1890			
Glu	Pro	Ile	Thr	Thr	Thr	Leu	Lys	Arg	Lys	Gln	Glu	Glu	Val	Ser



## SSCP Update Sequences.ST25

1895

1900

1905

Ala Ile Ile Ile Gln Arg Ala Tyr Arg Arg Tyr Leu Leu Lys Gln  
 1910 1915 1920

Lys Val Lys Lys Val Ser Ser Ile Tyr Lys Lys Asp Lys Gly Lys  
 1925 1930 1935

Glu Cys Asp Gly Thr Pro Ile Lys Glu Asp Thr Leu Ile Asp Lys  
 1940 1945 1950

Leu Asn Glu Asn Ser Thr Pro Glu Lys Thr Asp Met Thr Pro Ser  
 1955 1960 1965

Thr Thr Ser Pro Pro Ser Tyr Asp Ser Val Thr Lys Pro Glu Lys  
 1970 1975 1980

Glu Lys Phe Glu Lys Asp Lys Ser Glu Lys Glu Asp Lys Gly Lys  
 1985 1990 1995

Asp Ile Arg Glu Ser Lys Lys  
 2000 2005

<210> 88  
 <211> 468  
 <212> PRT  
 <213> Homo sapiens

<400> 88

Met Ala Ala Arg Gly Ser Gly Pro Arg Ala Leu Arg Leu Leu Leu Leu  
 1 5 10 15

Val Gln Leu Val Ala Gly Ala Leu Arg Ser Ser Arg Ala Arg Arg Ala  
 20 25 30

Ala Arg Arg Gly Leu Ser Glu Pro Ser Ser Ile Ala Lys His Glu Asp  
 35 40 45

Ser Leu Leu Lys Asp Leu Phe Gln Asp Tyr Glu Arg Trp Val Arg Pro  
 50 55 60

Val Glu His Leu Asn Asp Lys Ile Lys Ile Lys Phe Gly Leu Ala Ile  
 65 70 75 80

Ser Gln Leu Val Asp Val Asp Glu Lys Asn Gln Leu Met Thr Thr Asn  
 85 90 95

Val Trp Leu Lys Gln Glu Trp Ile Asp Val Lys Leu Arg Trp Asn Pro  
 100 105 110

Asp Asp Tyr Gly Gly Ile Lys Val Ile Arg Val Pro Ser Asp Ser Ser  
 115 120 125

## SSCP update Sequences.ST25

Trp Thr Pro Asp Ile Ile Leu Phe Asp Asn Ala Asp Gly Arg Phe Glu  
 130 135 140  
 Gly Thr Ser Thr Lys Thr Val Ile Arg Tyr Asn Gly Thr Val Thr Trp  
 145 150 155 160  
 Thr Pro Pro Ala Asn Tyr Lys Ser Ser Cys Thr Ile Asp Val Thr Phe  
 165 170 175  
 Phe Pro Phe Asp Leu Gln Asn Cys Ser Met Lys Phe Gly Ser Trp Thr  
 180 185 190  
 Tyr Asp Gly Ser Gln Val Asp Ile Ile Leu Glu Asp Gln Asp Val Asp  
 195 200 205  
 Lys Arg Asp Phe Phe Asp Asn Gly Glu Trp Glu Ile Val Ser Ala Thr  
 210 215 220  
 Gly Ser Lys Gly Asn Arg Thr Asp Ser Cys Cys Trp Tyr Pro Tyr Val  
 225 230 235 240  
 Thr Tyr Ser Phe Val Ile Lys Arg Leu Pro Leu Phe Tyr Thr Leu Phe  
 245 250 255  
 Leu Ile Ile Pro Cys Ile Gly Leu Ser Phe Leu Thr Val Leu Val Phe  
 260 265 270  
 Tyr Leu Pro Ser Asn Glu Gly Glu Lys Ile Cys Leu Cys Thr Ser Val  
 275 280 285  
 Leu Val Ser Leu Thr Val Phe Leu Leu Val Ile Glu Glu Ile Ile Pro  
 290 295 300  
 Ser Ser Ser Lys Val Ile Pro Leu Ile Gly Glu Tyr Leu Val Phe Thr  
 305 310 315 320  
 Met Ile Phe Val Thr Leu Ser Ile Met Val Thr Val Phe Ala Ile Asn  
 325 330 335  
 Ile His His Arg Ser Ser Ser Thr His Asn Ala Met Ala Pro Leu Val  
 340 345 350  
 Arg Lys Ile Phe Leu His Thr Leu Pro Lys Leu Leu Ser Met Arg Ser  
 355 360 365  
 His Val Asp Arg Tyr Phe Thr Gln Lys Glu Glu Thr Glu Ser Gly Ser  
 370 375 380  
 Gly Pro Lys Ser Ser Arg Asn Thr Leu Glu Ala Ala Leu Asp Ser Ile  
 385 390 395 400

## SSCP update Sequences.ST25

Arg Tyr Ile Thr Thr His Ile Met Lys Glu Asn Asp Val Arg Glu Val  
405 410 415

Val Glu Asp Trp Lys Phe Ile Ala Gln Val Leu Asp Arg Met Phe Leu  
420 425 430

Trp Thr Phe Leu Phe Val Ser Ile Val Gly Ser Leu Gly Leu Phe Val  
435 440 445

Pro Val Ile Tyr Lys Trp Ala Asn Ile Leu Ile Pro Val His Ile Gly  
450 455 460

Asn Ala Asn Lys  
465

<210> 89  
<211> 529  
<212> PRT  
<213> Homo sapiens

<400> 89

Met Gly Pro Ser Cys Pro Val Phe Leu Ser Phe Thr Lys Leu Ser Leu  
1 5 10 15

Trp Trp Leu Leu Leu Thr Pro Ala Gly Gly Glu Glu Ala Lys Arg Pro  
20 25 30

Pro Pro Arg Ala Pro Gly Asp Pro Leu Ser Ser Pro Ser Pro Thr Ala  
35 40 45

Leu Pro Gln Gly Gly Ser His Thr Glu Thr Glu Asp Arg Leu Phe Lys  
50 55 60

His Leu Phe Arg Gly Tyr Asn Arg Trp Ala Arg Pro Val Pro Asn Thr  
65 70 75 80

Ser Asp Val Val Ile Val Arg Phe Gly Leu Ser Ile Ala Gln Leu Ile  
85 90 95

Asp Val Asp Glu Lys Asn Gln Met Met Thr Thr Asn Val Trp Leu Lys  
100 105 110

Gln Glu Trp Ser Asp Tyr Lys Leu Arg Trp Asn Pro Thr Asp Phe Gly  
115 120 125

Asn Ile Thr Ser Leu Arg Val Pro Ser Glu Met Ile Trp Ile Pro Asp  
130 135 140

Ile Val Leu Tyr Asn Asn Ala Asp Gly Glu Phe Ala Val Thr His Met  
145 150 155 160

## SSCP Update Sequences.ST25

Thr Lys Ala His Leu Phe Ser Thr Gly Thr Val His Trp Val Pro Pro  
 165 170 175  
 Ala Ile Tyr Lys Ser Ser Cys Ser Ile Asp Val Thr Phe Phe Pro Phe  
 180 185 190  
 Asp Gln Gln Asn Cys Lys Met Lys Phe Gly Ser Trp Thr Tyr Asp Lys  
 195 200 205  
 Ala Lys Ile Asp Leu Glu Gln Met Glu Gln Thr Val Asp Leu Lys Asp  
 210 215 220  
 Tyr Trp Glu Ser Gly Glu Trp Ala Ile Val Asn Ala Thr Gly Thr Tyr  
 225 230 235 240  
 Asn Ser Lys Lys Tyr Asp Cys Cys Ala Glu Ile Tyr Pro Asp Val Thr  
 245 250 255  
 Tyr Ala Phe Val Ile Arg Arg Leu Pro Leu Phe Tyr Thr Ile Asn Leu  
 260 265 270  
 Ile Ile Pro Cys Leu Leu Ile Ser Cys Leu Thr Val Leu Val Phe Tyr  
 275 280 285  
 Leu Pro Ser Asp Cys Gly Glu Lys Ile Thr Leu Cys Ile Ser Val Leu  
 290 295 300  
 Leu Ser Leu Thr Val Phe Leu Leu Leu Ile Thr Glu Ile Ile Pro Ser  
 305 310 315 320  
 Thr Ser Leu Val Ile Pro Leu Ile Gly Glu Tyr Leu Leu Phe Thr Met  
 325 330 335  
 Ile Phe Val Thr Leu Ser Ile Val Ile Thr Val Phe Val Leu Asn Val  
 340 345 350  
 His His Arg Ser Pro Ser Thr His Thr Met Pro His Trp Val Arg Gly  
 355 360 365  
 Ala Leu Leu Gly Cys Val Pro Arg Trp Leu Leu Met Asn Arg Pro Pro  
 370 375 380  
 Pro Pro Val Glu Leu Cys His Pro Leu Arg Leu Lys Leu Ser Pro Ser  
 385 390 395 400  
 Tyr His Trp Leu Glu Ser Asn Val Asp Ala Glu Glu Arg Glu Val Val  
 405 410 415  
 Val Glu Glu Glu Asp Arg Trp Ala Cys Ala Gly His Val Ala Pro Ser  
 420 425 430

## SSCP Update Sequences.ST25

Val Gly Thr Leu Cys Ser His Gly His Leu His Ser Gly Ala Ser Gly  
 435 440 445

Pro Lys Ala Glu Ala Leu Leu Gln Glu Gly Glu Leu Leu Leu Ser Pro  
 450 455 460

His Met Gln Lys Ala Leu Glu Gly Val His Tyr Ile Ala Asp His Leu  
 465 470 475 480

Arg Ser Glu Asp Ala Asp Ser Ser Val Lys Glu Asp Trp Lys Tyr Val  
 485 490 495

Ala Met Val Ile Asp Arg Ile Phe Leu Trp Leu Phe Ile Ile Val Cys  
 500 505 510

Phe Leu Gly Thr Ile Gly Leu Phe Leu Pro Pro Phe Leu Ala Gly Met  
 515 520 525

Ile

<210> 90  
 <211> 505  
 <212> PRT  
 <213> Homo sapiens

<400> 90

Met Gly Ser Gly Pro Leu Ser Leu Pro Leu Ala Leu Ser Pro Pro Arg  
 1 5 10 15

Leu Leu Leu Leu Leu Leu Leu Ser Leu Leu Pro Val Ala Arg Ala Ser  
 20 25 30

Glu Ala Glu His His Leu Phe Glu Arg Leu Phe Glu Asp Tyr Asn Glu  
 35 40 45

Ile Ile Arg Pro Val Ala Asn Val Ser Asp Pro Val Ile Ile His Phe  
 50 55 60

Glu Val Ser Met Ser Gln Leu Val Lys Val Asp Glu Val Asn Gln Ile  
 65 70 75 80

Met Glu Thr Asn Leu Trp Leu Lys Gln Ile Trp Asn Asp Tyr Lys Leu  
 85 90 95

Lys Trp Asn Pro Ser Asp Tyr Gly Gly Ala Glu Phe Met Arg Val Pro  
 100 105 110

Ala Gln Lys Ile Trp Lys Pro Asp Ile Val Leu Tyr Asn Asn Ala Val  
 115 120 125

## SSCP Update Sequences.ST25

Gly Asp Phe Gln Val Asp Asp Lys Thr Lys Ala Leu Leu Lys Tyr Thr  
 130 135 140  
 Gly Glu Val Thr Trp Ile Pro Pro Ala Ile Phe Lys Ser Ser Cys Lys  
 145 150 155 160  
 Ile Asp Val Thr Tyr Phe Pro Phe Asp Tyr Gln Asn Cys Thr Met Lys  
 165 170 175  
 Phe Gly Ser Trp Ser Tyr Asp Lys Ala Lys Ile Asp Leu Val Leu Ile  
 180 185 190  
 Gly Ser Ser Met Asn Leu Lys Asp Tyr Trp Glu Ser Gly Glu Trp Ala  
 195 200 205  
 Ile Ile Lys Ala Pro Gly Tyr Lys His Asp Ile Lys Tyr Asn Cys Cys  
 210 215 220  
 Glu Glu Ile Tyr Pro Asp Ile Thr Tyr Ser Leu Tyr Ile Arg Arg Leu  
 225 230 235 240  
 Pro Leu Phe Tyr Thr Ile Asn Leu Ile Ile Pro Cys Leu Leu Ile Ser  
 245 250 255  
 Phe Leu Thr Val Leu Val Phe Tyr Leu Pro Ser Asp Cys Gly Glu Lys  
 260 265 270  
 Val Thr Leu Cys Ile Ser Val Leu Leu Ser Leu Thr Val Phe Leu Leu  
 275 280 285  
 Val Ile Thr Glu Thr Ile Pro Ser Thr Ser Leu Val Ile Pro Leu Ile  
 290 295 300  
 Gly Glu Tyr Leu Leu Phe Thr Met Ile Phe Val Thr Leu Ser Ile Val  
 305 310 315 320  
 Ile Thr Val Phe Val Leu Asn Val His Tyr Arg Thr Pro Thr Thr His  
 325 330 335  
 Thr Met Pro Ser Trp Val Lys Thr Val Phe Leu Asn Leu Leu Pro Arg  
 340 345 350  
 Val Met Phe Met Thr Arg Pro Thr Ser Asn Glu Gly Asn Ala Gln Lys  
 355 360 365  
 Pro Arg Pro Leu Tyr Gly Ala Glu Leu Ser Asn Leu Asn Cys Phe Ser  
 370 375 380  
 Arg Ala Glu Ser Lys Gly Cys Lys Glu Gly Tyr Pro Cys Gln Asp Gly  
 385 390 395 400

## SSCP Update Sequences.ST25

Met Cys Gly Tyr Cys His His Arg Arg Ile Lys Ile Ser Asn Phe Ser  
 405 410 415

Ala Asn Leu Thr Arg Ser Ser Ser Ser Glu Ser Val Asp Ala Val Leu  
 420 425 430

Ser Leu Ser Ala Leu Ser Pro Glu Ile Lys Glu Ala Ile Gln Ser Val  
 435 440 445

Lys Tyr Ile Ala Glu Asn Met Lys Ala Gln Asn Glu Ala Lys Glu Ile  
 450 455 460

Gln Asp Asp Trp Lys Tyr Val Ala Met Val Ile Asp Arg Ile Phe Leu  
 465 470 475 480

Trp Val Phe Thr Leu Val Cys Ile Leu Gly Thr Ala Gly Leu Phe Leu  
 485 490 495

Gln Pro Leu Met Ala Arg Glu Asp Ala  
 500 505

<210> 91  
 <211> 118  
 <212> PRT  
 <213> Homo sapiens

<400> 91

Met Val Gln Lys Ser Arg Asn Gly Gly Val Tyr Pro Gly Pro Ser Gly  
 1 5 10 15

Glu Lys Lys Leu Lys Val Gly Phe Val Gly Leu Asp Pro Gly Ala Pro  
 20 25 30

Asp Ser Thr Arg Asp Gly Ala Leu Leu Ile Ala Gly Ser Glu Ala Pro  
 35 40 45

Lys Arg Gly Ser Ile Leu Ser Lys Pro Arg Ala Gly Gly Ala Gly Ala  
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Gly Lys Pro Pro Gln Ala Gln Arg Leu Leu Pro Gln Ala Ala Glu Phe  
 65 70 75 80

Pro Leu Gln Arg Ala Gly Ala Ala Ala Arg Leu Gly Val His Leu Pro  
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Phe His His Gln Gly Val  
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<210> 92

## SSCP Update Sequences.ST25

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 35 40 45  
 Lys Arg Gly Ser Ile Leu Ser Lys Pro Arg Ala Gly Gly Ala Gly Ala  
 50 55 60  
 Gly Lys Pro Pro Lys Arg Asn Ala Phe Tyr Arg Lys Leu Gln Asn Phe  
 65 70 75 80  
 Leu Tyr Asn Val Leu Glu Arg Pro Arg Gly Trp Ala Phe Ile Tyr His  
 85 90 95  
 Ala Tyr Val Phe Leu Leu Val Phe Ser Cys Leu Val Leu Ser Val Phe  
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 Ser Thr Ile Lys Glu Tyr Glu Lys Ser Ser Glu Gly Ala Leu Tyr Ile  
 115 120 125  
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 130 135 140  
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 Val Tyr Ala His Ser Lys Glu Leu Val Thr Ala Trp Tyr Ile Gly Phe  
 225 230 235 240  
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 245 250 255



Glu Asn Asp His Phe Asp Thr Tyr Ala Asp Ala Leu Trp Trp Gly Leu  
260 265 270

Ile Thr Leu Thr Thr Ile Gly Tyr Gly Asp Lys Tyr Pro Gln Thr Trp  
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Asn Gly Arg Leu Leu Ala Ala Thr Phe Thr Leu Ile Gly Val Ser Phe  
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Phe Ala Leu Pro Ala Gly Ile Leu Gly Ser Gly Phe Ala Leu Lys Val  
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Gln Glu Gln His Arg Gln Lys His Phe Glu Lys Arg Arg Asn Pro Ala  
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Ala Gly Leu Ile Gln Ser Ala Trp Arg Phe Tyr Ala Thr Asn Leu Ser  
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Gly Thr Asp Leu His Ser Thr Trp Gln Tyr Tyr Glu Arg Thr Val Thr  
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Val Pro Met Tyr Ser Ser Gln Thr Gln Thr Tyr Gly Ala Ser Arg Leu  
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Ile Pro Pro Leu Asn Gln Leu Glu Leu Leu Arg Asn Leu Lys Ser Lys  
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Ala Ala Lys Gly Lys Gly Ser Pro Gln Ala Gln Thr Val Arg Arg Ser  
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Pro Ser Ala Asp Gln Ser Leu Glu Asp Ser Pro Ser Lys Val Pro Lys  
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465 470 475 480

Lys Gly Ala Ala Ser Arg Gln Asn Ser Glu Glu Ala Ser Leu Pro Gly  
485 490 495

Glu Asp Ile Val Asp Asp Lys Ser Cys Pro Cys Glu Phe Val Thr Glu  
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Asp Leu Thr Pro Gly Leu Lys Val Ser Ile Arg Ala Val Cys Val Met  
515 520 525

## SSCP Update Sequences.ST25

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 Arg Gly Pro Ala Ile Thr Asp Lys Asp Arg Thr Lys Gly Pro Ala Glu  
 580 585 590  
 Ala Glu Leu Pro Glu Asp Pro Ser Met Met Gly Arg Leu Gly Lys Val  
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 Glu Lys Gln Val Leu Ser Met Glu Lys Lys Leu Asp Phe Leu Val Asn  
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 625 630 635 640  
 Phe Gly Ala Lys Glu Pro Glu Pro Ala Pro Pro Tyr His Ser Pro Glu  
 645 650 655  
 Asp Ser Arg Glu His Val Asp Arg His Gly Cys Ile Val Lys Ile Val  
 660 665 670  
 Arg Ser Ser Ser Ser Thr Gly Gln Lys Asn Phe Ser Ala Pro Pro Ala  
 675 680 685  
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 690 695 700  
 His Pro Arg Gln Gly His Gly Thr Ser Pro Val Gly Asp His Gly Ser  
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 Leu Val Arg Ile Pro Pro Pro Pro Ala His Glu Arg Ser Leu Ser Ala  
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 Tyr Gly Gly Gly Asn Arg Ala Ser Met Glu Phe Leu Arg Gln Glu Asp  
 740 745 750  
 Thr Pro Gly Cys Arg Pro Pro Glu Gly Thr Leu Arg Asp Ser Asp Thr  
 755 760 765  
 Ser Ile Ser Ile Pro Ser Val Asp His Glu Glu Leu Glu Arg Ser Phe  
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 Ser Gly Phe Ser Ile Ser Gln Ser Lys Glu Asn Leu Asp Ala Leu Asn  
 785 790 795 800

## SSCP Update Sequences.ST25

Ser Cys Tyr Ala Ala Val Ala Pro Cys Ala Lys Val Arg Pro Tyr Ile  
805 810 815

Ala Glu Gly Glu Ser Asp Thr Asp Ser Asp Leu Cys Thr Pro Cys Gly  
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835 840 845

Trp Ala Gly Pro Arg Lys  
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<210> 93  
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<212> PRT  
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<400> 93

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Asp Ser Thr Arg Asp Gly Ala Leu Leu Ile Ala Gly Ser Glu Ala Pro  
35 40 45

Lys Arg Gly Ser Ile Leu Ser Lys Pro Arg Ala Gly Gly Ala Gly Ala  
50 55 60

Gly Lys Pro Pro Lys Arg Asn Ala Phe Tyr Arg Lys Leu Gln Asn Phe  
65 70 75 80

Leu Tyr Asn Val Leu Glu Arg Pro Arg Gly Trp Ala Phe Ile Tyr His  
85 90 95

Ala Tyr Val Phe Leu Leu Val Phe Ser Cys Leu Val Leu Ser Val Phe  
100 105 110

Ser Thr Ile Lys Glu Tyr Glu Lys Ser Ser Glu Gly Ala Leu Tyr Ile  
115 120 125

Leu Glu Ile Val Thr Ile Val Val Phe Gly Val Glu Tyr Phe Val Arg  
130 135 140

Ile Trp Ala Ala Gly Cys Cys Cys Arg Tyr Arg Gly Trp Arg Gly Arg  
145 150 155 160

Leu Lys Phe Ala Arg Lys Pro Phe Cys Val Ile Asp Ile Met Val Leu  
165 170 175

## SSCP Update Sequences.ST25

Ile Ala Ser Ile Ala Val Leu Ala Ala Gly Ser Gln Gly Asn Val Phe  
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 Ala Thr Ser Ala Leu Arg Ser Leu Arg Phe Leu Gln Ile Leu Arg Met  
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 Ile Arg Met Asp Arg Arg Gly Gly Thr Trp Lys Leu Leu Gly Ser Val  
 210 215 220  
 Val Tyr Ala His Ser Lys Glu Leu Val Thr Ala Trp Tyr Ile Gly Phe  
 225 230 235 240  
 Leu Cys Leu Ile Leu Ala Ser Phe Leu Val Tyr Leu Ala Glu Lys Gly  
 245 250 255  
 Glu Asn Asp His Phe Asp Thr Tyr Ala Asp Ala Leu Trp Trp Gly Leu  
 260 265 270  
 Ile Thr Leu Thr Thr Ile Gly Tyr Gly Asp Lys Tyr Pro Gln Thr Trp  
 275 280 285  
 Asn Gly Arg Leu Leu Ala Ala Thr Phe Thr Leu Ile Gly Val Ser Phe  
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 Phe Ala Leu Pro Ala Gly Ile Leu Gly Ser Gly Phe Ala Leu Lys Val  
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 Gln Glu Gln His Arg Gln Lys His Phe Glu Lys Arg Arg Asn Pro Ala  
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 Ala Gly Leu Ile Gln Ser Ala Trp Arg Phe Tyr Ala Thr Asn Leu Ser  
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 Arg Thr Asp Leu His Ser Thr Trp Gln Tyr Tyr Glu Arg Thr Val Thr  
 355 360 365  
 Val Pro Met Tyr Ser Ser Gln Thr Gln Thr Tyr Gly Ala Ser Arg Leu  
 370 375 380  
 Ile Pro Pro Leu Asn Gln Leu Glu Leu Leu Arg Asn Leu Lys Ser Lys  
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<210> 94  
 <211> 854  
 <212> PRT

## SSCP Update Sequences.ST25

&lt;213&gt; Homo sapiens

&lt;400&gt; 94

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20      25      30
.
Asp Ser Thr Arg Asp Gly Ala Leu Leu Ile Ala Gly Ser Glu Ala Pro
35      40      45
.
Lys Arg Gly Ser Ile Leu Ser Lys Pro Arg Ala Gly Gly Ala Gly Ala
50      55      60
.
Gly Lys Pro Pro Lys Arg Asn Ala Phe Tyr Arg Lys Leu Gln Asn Phe
65      70      75      80
.
Leu Tyr Asn Val Leu Glu Arg Pro Arg Gly Trp Ala Phe Ile Tyr His
85      90      95
.
Ala Tyr Val Phe Leu Leu Val Phe Ser Cys Leu Val Leu Ser Val Phe
100      105      110
.
Ser Thr Ile Lys Glu Tyr Glu Lys Ser Ser Glu Gly Ala Leu Tyr Ile
115      120      125
.
Leu Glu Ile Val Thr Ile Val Val Phe Gly Val Glu Tyr Phe Val Arg
130      135      140
.
Ile Trp Ala Ala Gly Cys Cys Cys Arg Tyr Arg Gly Trp Arg Gly Arg
145      150      155      160
.
Leu Lys Phe Ala Arg Lys Pro Phe Cys Val Ile Asp Ile Met Val Leu
165      170      175
.
Ile Ala Ser Ile Ala Val Leu Ala Ala Gly Ser Gln Gly Asn Val Phe
180      185      190
.
Ala Thr Ser Ala Leu Arg Ser Leu Arg Phe Leu Gln Ile Leu Arg Met
195      200      205
.
Ile Arg Met Asp Arg Arg Gly Gly Thr Trp Lys Leu Leu Gly Ser Val
210      215      220
.
Val Tyr Ala His Ser Lys Glu Leu Val Thr Ala Trp Tyr Ile Gly Phe
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.
Leu Cys Leu Ile Leu Ala Ser Phe Leu Val Tyr Leu Ala Glu Lys Gly
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## SSCP Update Sequences.ST25

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 Asn Gly Arg Leu Leu Ala Ala Thr Phe Thr Leu Ile Gly Val Ser Phe  
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 305 310 315 320  
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 325 330 335  
 Ala Gly Leu Ile Gln Ser Ala Trp Arg Phe Tyr Ala Thr Asn Leu Ser  
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 355 360 365  
 Val Pro Met Tyr Ser Ser Gln Thr Gln Thr Tyr Gly Ala Ser Arg Leu  
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 385 390 395 400  
 Ser Gly Leu Ala Phe Arg Lys Asp Pro Pro Pro Glu Pro Ser Pro Ser  
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 Gln Lys Val Ser Leu Lys Asp Arg Val Phe Ser Ser Pro Arg Gly Val  
 420 425 430  
 Ala Ala Lys Gly Lys Gly Ser Pro Gln Ala Gln Thr Val Arg Arg Ser  
 435 440 445  
 Pro Ser Ala Asp Gln Ser Leu Glu Asp Ser Pro Ser Lys Val Pro Lys  
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 Ser Trp Ser Phe Gly Asp Arg Ser Arg Ala Arg Gln Ala Phe Arg Ile  
 465 470 475 480  
 Lys Gly Ala Ala Ser Arg Gln Asn Ser Glu Glu Ala Ser Leu Pro Gly  
 485 490 495  
 Glu Asp Ile Val Asp Asp Lys Ser Cys Pro Cys Glu Phe Val Thr Glu  
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 Asp Leu Thr Pro Gly Leu Lys Val Ser Ile Arg Ala Val Cys Val Met  
 515 520 525

## SSCP Update Sequences.ST25

Arg Phe Leu Val Ser Lys Arg Lys Phe Lys Glu Ser Leu Arg Pro Tyr  
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 Asp Val Met Asp Val Ile Glu Gln Tyr Ser Ala Gly His Leu Asp Met  
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 Leu Ser Arg Ile Lys Ser Leu Gln Ser Ser Val Asp Gln Ile Val Gly  
 565 570 575  
 Arg Gly Pro Ala Ile Thr Asp Lys Asp Arg Thr Lys Gly Pro Ala Glu  
 580 585 590  
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 595 600 605  
 Glu Lys Gln Val Leu Ser Met Glu Lys Lys Leu Asp Phe Leu Val Asn  
 610 615 620  
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 645 650 655  
 Asp Ser Arg Glu His Val Asp Arg His Gly Cys Ile Val Lys Ile Val  
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 675 680 685  
 Ala Pro Pro Val Gln Cys Pro Pro Ser Thr Ser Trp Gln Pro Gln Ser  
 690 695 700  
 His Pro Arg Gln Gly His Gly Thr Ser Pro Val Gly Asp His Gly Ser  
 705 710 715 720  
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 Tyr Gly Gly Gly Asn Arg Ala Ser Met Glu Phe Leu Arg Gln Glu Asp  
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 Thr Pro Gly Cys Arg Pro Pro Glu Gly Thr Leu Arg Asp Ser Asp Thr  
 755 760 765  
 Ser Ile Ser Ile Pro Ser Val Asp His Glu Glu Leu Glu Arg Ser Phe  
 770 775 780  
 Ser Gly Phe Ser Ile Ser Gln Ser Lys Glu Asn Leu Asp Ala Leu Asn  
 785 790 795 800

## SSCP Update Sequences.ST25

Ser Cys Tyr Ala Ala Val Ala Pro Cys Ala Lys Val Arg Pro Tyr Ile  
805 810 815

Ala Glu Gly Glu Ser Asp Thr Asp Ser Asp Leu Cys Thr Pro Cys Gly  
820 825 830

Pro Pro Pro Arg Ser Ala Thr Gly Glu Gly Pro Phe Gly Asp Val Gly  
835 840 845

Trp Ala Gly Pro Arg Lys  
850

<210> 95  
<211> 854  
<212> PRT  
<213> Homo sapiens

<400> 95

Met Val Gln Lys Ser Arg Asn Gly Gly Val Tyr Pro Gly Pro Ser Gly  
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Asp Ser Thr Arg Asp Gly Ala Leu Leu Ile Ala Gly Ser Glu Ala Pro  
35 40 45

Lys Arg Gly Ser Ile Leu Ser Lys Pro Arg Ala Gly Gly Ala Gly Ala  
50 55 60

Gly Lys Pro Pro Lys Arg Asn Ala Phe Tyr Arg Lys Leu Gln Asn Phe  
65 70 75 80

Leu Tyr Asn Val Leu Glu Arg Pro Arg Gly Trp Ala Phe Ile Tyr His  
85 90 95

Ala Tyr Val Phe Leu Leu Val Phe Ser Cys Leu Val Leu Ser Val Phe  
100 105 110

Ser Thr Ile Lys Glu Tyr Glu Lys Ser Ser Glu Gly Ala Leu Tyr Ile  
115 120 125

Leu Glu Ile Val Thr Ile Val Val Phe Gly Val Glu Tyr Phe Val Arg  
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Ile Trp Ala Ala Gly Cys Cys Cys Arg Tyr Arg Gly Trp Arg Gly Arg  
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Leu Lys Phe Ala Arg Lys Pro Phe Cys Val Ile Asp Ile Met Val Leu  
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Ile Ala Ser Ile Ala Val Leu Ala Ala Gly Ser Gln Gly Asn Val Phe  
Page 230



SSCP Update Sequences.ST25  
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180

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225 230 235 240Leu Cys Leu Ile Leu Ala Ser Phe Leu Val Tyr Leu Ala Glu Lys Gly  
245 250 255Glu Asn Asp His Phe Asp Thr Tyr Ala Asp Ala Leu Trp Trp Gly Leu  
260 265 270Ile Thr Leu Thr Thr Ile Gly Tyr Gly Asp Lys Tyr Pro Gln Thr Trp  
275 280 285Asn Gly Arg Leu Leu Ala Ala Thr Phe Thr Leu Ile Gly Val Ser Phe  
290 295 300Phe Ala Leu Pro Ala Gly Ile Leu Gly Ser Gly Phe Ala Leu Lys Val  
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Page 231

## SSCP Update Sequences.ST25

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455

460

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Lys Gly Ala Ala Ser Arg Gln Asn Ser Glu Glu Ala Ser Leu Pro Gly  
 485 490 495

Glu Asp Ile Val Asp Asp Lys Ser Cys Pro Cys Glu Phe Val Thr Glu  
 500 505 510

Asp Leu Thr Pro Gly Leu Lys Val Ser Ile Arg Ala Val Cys Val Met  
 515 520 525

Arg Phe Leu Val Ser Lys Arg Lys Phe Lys Glu Ser Leu Arg Pro Tyr  
 530 535 540

Asp Val Met Asp Val Ile Glu Gln Tyr Ser Ala Gly His Leu Asp Met  
 545 550 555 560

Leu Ser Arg Ile Lys Ser Leu Gln Ser Arg Val Asp Gln Ile Val Gly  
 565 570 575

Arg Gly Pro Ala Ile Thr Asp Lys Asp Arg Thr Lys Gly Pro Ala Glu  
 580 585 590

Ala Glu Leu Pro Glu Asp Pro Ser Met Met Gly Arg Leu Gly Lys Val  
 595 600 605

Glu Lys Gln Val Leu Ser Met Glu Lys Lys Arg Asp Phe Leu Val Asn  
 610 615 620

Ile Tyr Met Gln Arg Met Gly Ile Pro Pro Thr Glu Thr Glu Ala Tyr  
 625 630 635 640

Phe Gly Ala Lys Glu Pro Glu Pro Ala Pro Pro Tyr His Ser Pro Glu  
 645 650 655

Asp Ser Arg Glu His Val Asp Arg His Gly Cys Ile Val Lys Ile Val  
 660 665 670

Arg Ser Ser Ser Ser Thr Gly Gln Lys Asn Phe Ser Ala Pro Pro Ala  
 675 680 685

Ala Pro Pro Val Gln Cys Pro Pro Ser Thr Ser Trp Gln Pro Gln Ser  
 690 695 700

His Pro Arg Gln Gly His Gly Thr Ser Pro Val Gly Asp His Gly Ser  
 705 710 715 720

Leu Val Arg Ile Pro Pro Pro Pro Ala His Glu Arg Ser Leu Ser Ala

## SSCP Update Sequences.ST25

725

730

735

Tyr Gly Gly Gly Asn Arg Ala Ser Met Glu Phe Leu Arg Gln Glu Asp  
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Thr Pro Gly Cys Arg Pro Pro Glu Gly Thr Leu Arg Asp Ser Asp Thr  
 755 760 765

Ser Ile Ser Ile Pro Ser Val Asp His Glu Glu Leu Glu Arg Ser Phe  
 770 775 780

Ser Gly Phe Ser Ile Ser Gln Ser Lys Glu Asn Leu Asp Ala Leu Asn  
 785 790 795 800

Ser Cys Tyr Ala Ala Val Ala Pro Cys Ala Lys Val Arg Pro Tyr Ile  
 805 810 815

Ala Glu Gly Glu Ser Asp Thr Asp Ser Asp Leu Cys Thr Pro Cys Gly  
 820 825 830

Pro Pro Pro Arg Ser Ala Thr Gly Glu Gly Pro Phe Gly Asp Val Gly  
 835 840 845

Trp Ala Gly Pro Arg Lys  
 850

# INTERNATIONAL SEARCH REPORT

International application No.  
**PCT/AU2004/001051**

## A. CLASSIFICATION OF SUBJECT MATTER

Int. Cl. <sup>7</sup>: C12Q 1/68, C12N 15/01, A61K 39/395 CO7K14/47

According to International Patent Classification (IPC) or to both national classification and IPC

## B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

See electronic databases

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

See electronic databases

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)

WPIDS, CA Medline. SCN1A, polymorphism/mutation/SNP, epilepsy/disease/febrile seizure

## C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
A	Fujiwara T et al. Mutations of sodium channel $\alpha$ subunit type 1 (SCN1A) in intractable childhood epilepsies with frequent generalized tonic-clonic seizures. Brain, 2003. 126: 531-546	
A	Nabbout R et al. Spectrum of SCN1A mutations in severe myoclonic epilepsy of infancy. Neurology, 2003 Jun 24. 60(12):1961-7.	
A	WO 2003/008574 A1 (BIONOMICS LIMITED) 30 January 2003	
A	WO 2002/06521 A1 (BIONOMICS LIMITED) 24 January 2002	
A	WO 2002/50096 A1 ((BIONOMICS LIMITED) 27 June 2002	

☐ Further documents are listed in the continuation of Box C

☒ See patent family annex

\* Special categories of cited documents:

"A" document defining the general state of the art which is not considered to be of particular relevance

"E" earlier application or patent but published on or after the international filing date

"L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)

"O" document referring to an oral disclosure, use, exhibition or other means

"P" document published prior to the international filing date but later than the priority date claimed

"T" later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention

"X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone

"Y" document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art

"&" document member of the same patent family

Date of the actual completion of the international search  
**28 September 2004**

Date of mailing of the international search report

**7 OCT 2004**

Name and mailing address of the ISA/AU

AUSTRALIAN PATENT OFFICE  
PO BOX 200, WODEN ACT 2606, AUSTRALIA  
E-mail address: pct@ipaustalia.gov.au  
Facsimile No. (02) 6285 3929

Authorized officer

**Gillian Allen**

Telephone No : (02) 6283 2266

# INTERNATIONAL SEARCH REPORT

International application No.

PCT/AU2004/001051

## Box No. II Observations where certain claims were found unsearchable (Continuation of item 2 of first sheet)

This international search report has not been established in respect of certain claims under Article 17(2)(a) for the following reasons:

1. ☐ Claims Nos.:  
because they relate to subject matter not required to be searched by this Authority, namely:
2. ☒ Claims No 65 and 66  
because they relate to parts of the international application that do not comply with the prescribed requirements to such an extent that no meaningful international search can be carried out, specifically:  
The scope of these claims is so unclear that no meaningful search can be performed.
3. ☐ Claims Nos.:  
because they are dependent claims and are not drafted in accordance with the second and third sentences of Rule 6.4(a)

## Box No. III Observations where unity of invention is lacking (Continuation of item 3 of first sheet)

This International Searching Authority found multiple inventions in this international application, as follows:

The ISA found that the claims were directed to multiple invention

See Supplemental Box III for details

1. ☐ As all required additional search fees were timely paid by the applicant, this international search report covers all searchable claims.
2. ☐ As all searchable claims could be searched without effort justifying an additional fee, this Authority did not invite payment of any additional fee.
3. ☐ As only some of the required additional search fees were timely paid by the applicant, this international search report covers only those claims for which fees were paid, specifically claims Nos.:
4. ☒ No required additional search fees were timely paid by the applicant. Consequently, this international search report is restricted to the invention first mentioned in the claims; it is covered by:  
Claims 1-16 19-27, 29-64, 67-85 in so far as they are directed to polymorphisms in SCN1A

### Remark on Protest

- ☐ The additional search fees were accompanied by the applicant's protest.
- ☐ No protest accompanied the payment of additional search fees.

## INTERNATIONAL SEARCH REPORT

International application No.

PCT/AU2004/001051

### Supplemental Box

(To be used when the space in any of Boxes I to VIII is not sufficient)

#### Continuation of Box No: III

The present claims are to 72 different mutations in 18 different ion channel genes, the mutant genes and their encoded polypeptides and antibodies thereto, and to uses of these in diagnosis or therapy.

The unifying feature of the claimed inventions is a disease-associated mutation of an ion channel gene. However, ion channel disease-associated mutations are known for every one of the ion channels of the claims, ie SCN, CHRN, KCQN, and GABR.

Therefore, since the unifying feature of the different mutations is not novel, it cannot be accepted as a special technical feature that would unite the claims.

There are therefore 72 separate inventions claimed.

However, this office believes that all claimed mutations of any one of the claimed genes could be searched without undue effort, and has chose to search the claims in so far as they are directed to polymorphisms of SCN1A

## INTERNATIONAL SEARCH REPORT

International application No.

**PCT/AU2004/001051**

This Annex lists the known "A" publication level patent family members relating to the patent documents cited in the above-mentioned international search report. The Australian Patent Office is in no way liable for these particulars which are merely given for the purpose of information.

Patent Document Cited in Search Report	Patent Family Member
WO 2003/008574	CA 2454073 EP 1407013
WO 2002/06521	AU 200172218
WO 2002/50096	AU 200216826 EP 1351968 US 2004110706 JP 2004515252T

Due to data integration issues this family listing may not include 10 digit Australian applications filed since May 2001.

END OF ANNEX